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Air Quality Criteria for Ozone and Other Photochemical Oxidants

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This document consolidates and assesses current knowledge regarding the origin of ozone and other photochemical oxidants and discusses their effect on health, vegetation, certain ecosystems, and materials. This document summarizes current data on the effects of oxidant/ozone in the ambient air on man, vegetation, and ecosystems. The effects that have been observed will form the scientific basis for supporting the present National Ambient Air Quality Standard of 160 micrograms/cu m (0.08 ppm) or a revised standard. Although nitrogen dioxide is considered one of the photochemical oxidants, oxides of nitrogen are the subject of a separate report and are therefore discussed in this document only as they participate in the formation and reactions of other photochemical oxidants. Hydrocarbons and other organics are important air pollutants because they too are precursors of other compounds formed in the atmospheric photochemical system. In this document, toxic organics are considered only with respect to eye irritation. The studies and data cited constitute the best ava, ...

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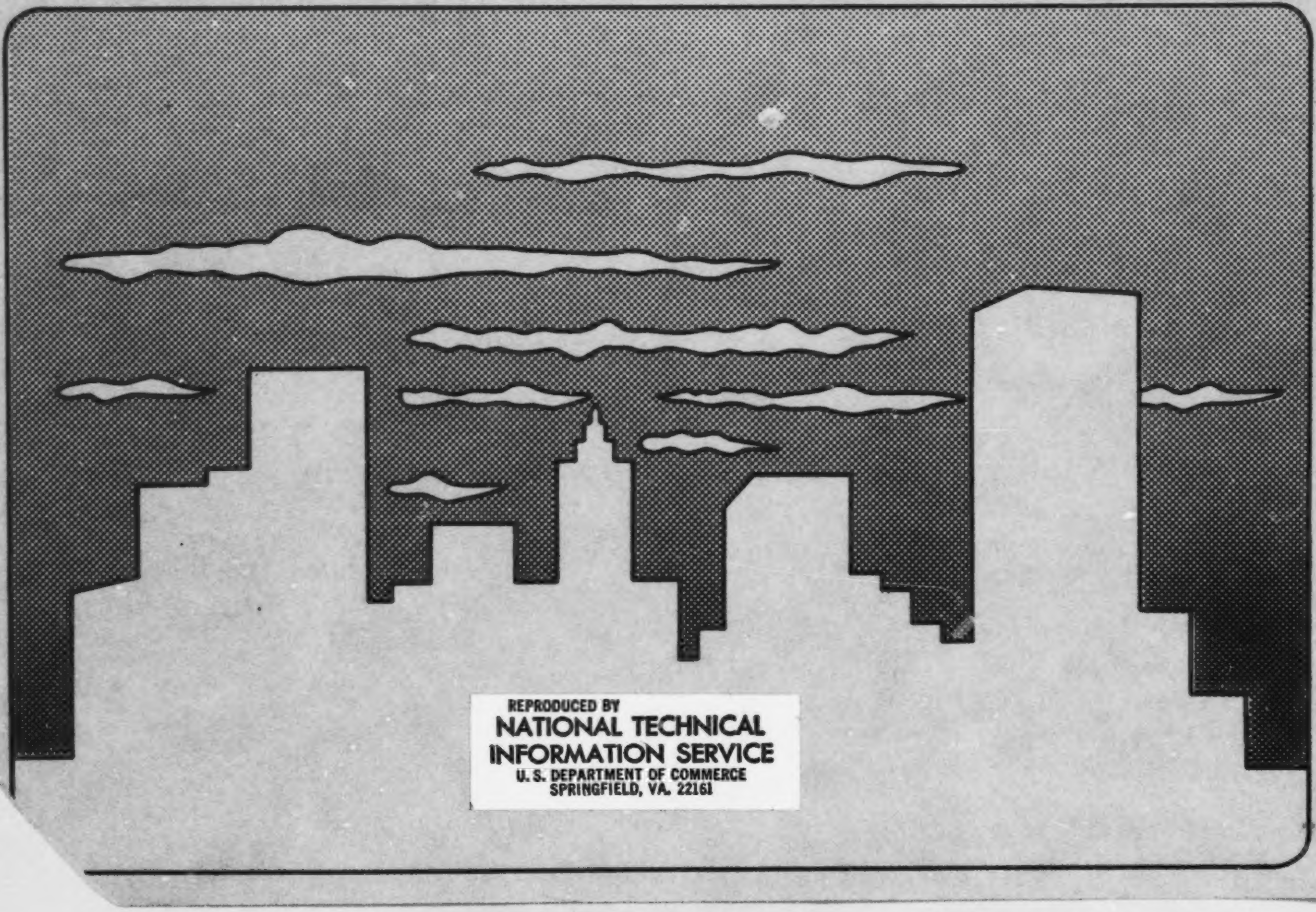

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AIR QUALITY CRITERIA FOR OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FOREWORD

This document has been prepared pursuant to Section 108(c) of the Clean Air Act, as amended, which requires that the Administrator from time to time review and, as appropriate, modify and reissue criteria issued pursuant to Section 108(a). Air quality criteria are required by Section 108(a) to reflect accurately the latest scientific information useful in indicating the kind and extent of all identifiable effects on public health and welfare that may be expected from the presence of a pollutant in the ambient air in varying quantities.

The original criteria document for photochemical oxidants, AP-63, was issued in 1970. Since that time, new information has been developed, and this document represents the modification and reissuance of the air quality criteria for photochemical oxidants.

The regulatory purpose of these criteria is to serve as the basis for national ambient air quality standards promulgated by the Administrator under Section 109 of the Clean Air Act, as amended. Accordingly, as provided by Section 109(d), the Administrator has reviewed the national ambient air quality standards for photochemical oxidants based on these revised criteria and is proposing appropriate action with respect to those standards concurrently with the issuance of this document.

The Agency is pleased to acknowledge the efforts and contributions of all persons and groups who have contributed to this document as participating authors or reviewers. In the last analysis, however, the Environmental Protection Agency is responsible for its content.

DOUGLAS M. COSTLE
Administrator
U.S. Environmental Protection Agency

micrograms/cu f m

PREFACE

This document consolidates and assesses current knowledge regarding the origin of ozone and other photochemical oxidants and discusses their effect on health, vegetation, certain ecosystems, and materials.

Photochemical oxidants are products of atmospheric reactions involving hydrocarbons (HC), nitrogen oxides (NO_x), oxygen, and sunlight. Oxidants consist mostly of ozone, nitrogen dioxide (NO_2), and peroxyacetylnitrate (PAN), with smaller amounts of other peroxyacylnitrates, other oxy- and peroxy-compounds, formic and nitric acid, and formaldehyde. Usually referred to collectively as "oxidants," they originate mainly from human activities that produce HC and NO_x emissions.

This document summarizes current data on the effects of oxidant/ozone in the ambient air on man, vegetation, and ecosystems. The effects that have been observed will form the scientific basis for supporting the present National Ambient Air Quality Standard of 160 $\mu g/m^3$ (0.08 ppm) or a revised standard.

Although nitrogen dioxide is considered one of the photochemical oxidants, oxides of nitrogen are the subject of a separate report and are therefore discussed in this document only as they participate in the formation and reactions of other photochemical oxidants. Hydrocarbons and other organics are important air pollutants because they too are precursors of other compounds formed in the atmospheric photochemical system. In this document, toxic organics are considered only with respect to eye irritation.

The studies and data cited constitute the best available basis for specific standards aimed at protecting human health and the environment from photochemical oxidants in ambient air.

CONTRIBUTORS AND REVIEWERS

The following individuals participated in the preparation of this document.

Authors

Dr. J. H. B. Garner
Project Officer for Ozone and Other
Photochemical Oxidants
Environmental Criteria and Assessment Office
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Frank Black
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Robert Chapman
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Kenneth L. Demerjian
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Basil Dimitriades
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Donald E. Gardner
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Ms. Judith A. Graham
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Fred H. Haynie
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Milan J. Hazucha
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. John H. Margeson
Environmental Monitoring and Support Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Frederick J. Miller
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. Richard J. Paur
Environmental Monitoring and Support Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Larry Purdue
Environmental Monitoring and Support Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Robert K. Stevens
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Environmental Protection Agency Review Group

Dr. J. H. B. Garner, Chairman
Environmental Criteria and Assessment Office
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. John Clements
Environmental Monitoring and Support Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Robert Fankhauser
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Thomas B. Feagans
Strategies and Air Standards Division
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Charles R. Hosler
Environmental Sciences Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Michael H. Jones
Strategies and Air Standards Division
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. John H. Knelson
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Mr. Ted Ripberger
Industrial Environmental Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, N.C.

Dr. David Tingey
Ecological Effects Division
U.S. Environmental Protection Agency
Corvallis, Ore.

Dr. David Weber
Ecological Effects Division
U.S. Environmental Protection Agency
Corvallis, Ore.

Dr. Raymond Wilhour
Ecological Effects Division
U.S. Environmental Protection Agency
Corvallis, Ore.

Consultants

Dr. Stephen M. Ayres
Chairman, Department of Internal Medicine
St. Louis University School of Medicine
St. Louis, Mo.

Dr. David V. Bates
Dean, School of Medicine
University of British Columbia
Vancouver, B.C., Canada

Dr. T. Timothy Crocker
Chairman, Department of Community and
  Environmental Medicine
University of California College of Medicine
Irvine, Calif.

Dr. Richard Ehrlich
Director, Life Sciences Division
Illinois Institute of Technology Research Institute
Chicago, Ill.

Dr. Gustave Freeman
Director, Department of Medical Sciences
Life Sciences Division
Stanford Research Institute
Menlo Park, Calif.

Dr. Bernard D. Goldstein
Associate Professor, Department of Medicine
  and Department of Environmental Medicine
New York University School of Medicine
New York, N.Y.

Dr. John R. Goldsmith
Environmental Epidemiology Unit
California State Department of Public Health
Sacramento, Calif.

Dr. Jack D. Hackney
Chief, Environmental Health Laboratories
Associate Professor of Medicine
Rancho Los Amigos Hospital Campus
  of the University of Southern California
Downey, Calif.

Dr. Sagar V. Krupa
Assistant Professor, Department of Plant Pathology
University of Minnesota
St. Paul, Minn.

Dr. Daniel B. Menzel
Associate Professor of Pharmacology and Medicine
  and Director, Laboratory of Environmental Pharmacology
  and Toxicology
Duke University Medical Center
Durham, N.C.

Dr. Carl M. Shy
Institute of Environmental Health
University of North Carolina
Chapel Hill, N.C.

Dr. Boyd R. Strain
Professor, Department of Botany
Duke University
Durham, N.C.

Technical Assistance

Environmental Protection Agency
Research Triangle Park, N.C.

Ms. F. Vandiver Duffield
Mr. Douglas B. Fennell
Mr. R. Wayne Fulford
Mr. Allen Hoyt
Ms. Evelynne R. Rash
Ms. Beverly A. Tilton

DOT Systems, Inc.
Vienna, Va.

Mr. Frank Broadwell
Dr. John Preston
Dr. Carolyn Pyrek
Mr. Arthur Willey

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ABBREVIATIONS AND SYMBOLS

Å	Ångstrom
AaD_{O_2}	Alveolar-arterial gradient
AChE	Acetylcholinesterase
AIChE	American Institute of Chemical Engineers
AM	Alveolar macrophages
APCD	Air pollution control district
AQSM	Air quality simulation model
Ar	Argon
A_2O_3	Arsenous oxide
ATPase	Adenosine triphosphatase
Avg	Average
BAKI	Potassium iodide solution acidified with boric acid
bd ft	Board foot
^7Be	Radioisotope of beryllium
Br_2	Bromine
^{B}scat	Extinction coefficient due to scatter by aerosols
°C	Degrees Celsius
14_c	Radioisotope of carbon
CAMP	Community Air Monitoring Program
CARB	California Air Resources Board
C_1-C_3	Hydrocarbons containing one to three carbon atoms
C_1-C_4	Hydrocarbons containing one to four carbon atoms
C_{2+}	Hydrocarbons containing more than two carbon atoms
C_2H_2	Acetylene
C_{4+}	Hydrocarbons containing more than four carbon atoms
$C_{4,5}$	Hydrocarbons containing four or five carbon atoms
C_4-C_6	Hydrocarbons containing four to six carbon atoms
C_5-C_{10}	Hydrocarbons containing five to ten carbon atoms
C_6H_5	Benzyl
$C_6H_5COO_2NO_2$	Peroxybenzoylnitrate (PB_zN)
CC	Closing capacity
C_{dyn}	Dynamic compliance
$CH(CH_3)CHO$	Propionaldehyde
CH_2CHCHO	Acrolein
CH_2CH_2	Ethylene
CH_2CO	Ketene
CH_2O	Formaldehyde; also HCHO and H_2CO
CH_3	Methyl group
CH_3CHO	Acetaldehyde
$CH_3COO_2NO_2$	Peroxyacetylnitrate (PAN)
CH_4	Methane
Chem	Chemiluminescence
Cl_2	Chlorine

cm	Centimeter
cm^3	Cubic centimeter
CNS	Central nervous system
CO	Carbon monoxide
CO_2	Carbon dioxide
Coh	Coefficient of haze
COMT	Catechol-*o*-methyltransferase
conc.	Concentration
COOH	Carboxyl group
$COONO_2$	Peroxynitrate group
Cro_3	Chromium trioxide (chromic anhydride, chromic acid)
C_{st}	Static lung compliance
Cyt. P_{450}	Cytochrome P_{450}
2,4-D	2,4-Dichlorophenoxyacetic acid
dbh	Diameter breast height
DEN	Diethylnitrosamine
DIFKIN	Diffusion Kinetics Model
DL_{co}	Carbon monoxide diffusing capacity of lung
DNA	Deoxyribonucleic acid
DOLA	Downtown Los Angeles
DW	Dry weight
EA	Environment Agency (Japan)
EC	Electron capture detection
EH	Humidity during exposure
EKMA	Empirical Kinetic Modeling Approach
EPA	U. S. Environmental Protection Agency
ESR	Electron spin resonance
Et	Ethyl
F_1	First filial generation
F_2	Second filial generation
°F	Degrees Fahrenheit
FEF 25-50%	Mean expiratory flow during middle half of FVC
FEF 25-75%	Maximal midexpiratory flow rate
FEF 50%	Instantaneous expiratory flow at 50% of FVC
FEF_{max}	Peak expiratory flow
Fe_2O_3	Ferric oxide
FEV	Forced expiratory volume
$FEV_{0.75}$	0.75-second forced expiratory flow
$FEV_{1.0}$	1-second forced expiratory flow
$FEV_{3.0}$	3-second forced expiratory flow
FID	Flame ionization detection (or detector)
FRC	Functional residual capacity
FS	Fourier-transform spectroscopy; also FT
FT	Fourier-transform spectroscopy; also FS
ft	Foot
ft^2	Square foot
ft-c	Foot-candle
FVC	Forced vital capacity
g	Gram
Gaw/Vtg	Specific conductance
GC	Gas chromatography
GDI	Gas distribution index
gf	Emission growth factor
GH	Humidity during growth

GLC	Gas-liquid chromatography
GM	General Motors Corporation
G-6-P	Glucose-6-phosphate
G-6-PD	Glucose-6-phosphate dehydrogenase
GPT	Gas-phase titration
GSC	Gas-solid chromatography
GSH	Reduced glutathione
GSSG	Oxidized glutathione
GSSRase	Glutathione reductase
H	Hydrogen
H^+	Hydrogen ion
^3H	Tritium
ha	Hectare
HC	Hydrocarbons
HCHO	Formaldehyde; also CH_2O and H_2CO
HCOOH	Formic acid
He	Helium
Hg	Mercury
HI	Hemagglutination inhibition
HMP	Hexose monophosphate
HNO_2	Nitrous acid; also HONO
HNO_3	Nitric acid; also $HONO_2$
HO	Hydroxyl group; also OH
HO_2	Hydropheroxy radical
HONO	Nitrous acid
$HONO_2$	Nitric acid
HO_2NO	Pernitrous acid
HO_2NO_2	Pernitric acid; also $HOONO_2$
$HOONO_2$	Pernitric acid
hr	Hour
$h\nu$	Photon
H_2CO	Formaldehyde; also HCHO and CH_2O
H_2O	Water
H_2O_2	Hydrogen peroxide
H_2S	Hydrogen sulfide
H_2SO_4	Sulfuric acid
I	Intensity of sunlight
I_2	Iodine
I_3^-	Triiodide ion
131_I	Radioisotope of iodine
in.	Inch
IR	Infrared
J_λ	Actinic irradiance
k	Rate constant; or, dissociation constant
K	Potassium
K^+	Potassium ion
KBr	Potassium bromide
kg	Kilogram
K_H	Horizontal eddy diffusivity coefficient
KI	Potassium iodide
K_i	Photodissociation rate constant
km	Kilometer
kPa	Kilopascal
kV	Kilovolt

K_v	Vertical eddy diffusivity coefficient
LA	Los Angeles
LARPP	Los Angeles Reactive Pollutant Project
lb	Pound
LD_{50}	Dose lethal to 50% of recipients
LDH	Lactic acid dehydrogenase
LIRAQ	Livermore Regional Air Quality (Model)
l/min	Liters per minute
ln	Natural logarithm
LT_{50}	Time in which 50% of recipients of stated dose died
lx	Lux
M	Third body (in a reaction)
m	Meter
m	Meta
m^2	Square meter
m^3	Cubic meter
MAO	Monoamine oxidase
max	Maximum
mb	Millibar
MBC	Maximum breathing capacity
MCP	McKee, Childers, and Parr (test)
Me	Methyl
MEFR	Maximum expiratory flow rate
mg	Milligram
mi	Mile
mi^2	Square mile
ml	milliliter
min	Minute
min.	Minimum
mm	Millimeter
MMC	Mean meridional circulation
MMEF	Mid-maximal expiratory flow rate
mmol	Millimole
MnO_2	Manganese dioxide
mo	Month
mph	Miles per hour
MS	Mass spectroscopy
n	Normal
N_2	Nitrogen
ΔN_2	Single breath N_2 alveolar plateau
n/a	Not applicable
NAD	Nicotinamide adenine dinucleotide
NAAQS	National Ambient Air Quality Standard
NADB	National Air Data Bank
NADH	Reduced nicotinamide adenine dinucleotide
$NADP^+$	Nicotinamide adenine dinucleotide phosphate
NADPH	Reduced nicotinamide adenine dinucleotide phosphate
NAS	National Academy of Sciences
NATO/CCMS	North Atlantic Treaty Organization/Committee on Challenges of Modern Society
NBKI	Neutral buffered potassium iodide
NBS	National Bureau of Standards
ND	No data; or, not detectable

Ne	Neon
NEDS	National Emissions Data System
NH_3	Ammonia
nm	Nanometer
NMHC	Nonmethane hydrocarbons
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_x	Nitrogen oxides
N_2O	Nitrous oxide
N_2O_5	Nitrogen pentoxide
NPSH	Nonprotein sulfhydryls
o	Ortho
O	Atomic oxygen
O_2	Oxygen
O_3	Ozone
OAQPS	Office of Air Quality Planning and Standards, EPA
O.D.	Orthostatic dysregulation
$O(^1D)$ or $O(^1\Delta)$	Excited atomic oxygen
OH	Hydroxyl group; also HO
OH°	Hydroxyl radical
O_3.Olefin	Ozonides
$O(^3P)$	Ground-state atomic oxygen
O_x	Photochemical oxidants
P	Probability (of observed results occurring by chance)
p	Para
32_P	Radioisotope of phosphorus
PAN	Peroxyacetylnitrate
Pa_{O_2}	Arterial oxygen tension
PAQ	Present air quality
PBN	Peroxybutyrylnitrate
PB_zN	Peroxybenzoylnitrate
6-PG	6-phosphogluconate
6-P-GD	6-phosphogluconate dehydrogenase
pH	Measure of acidity (negative logarithm of hydrogen ion concentration)
$P_{iso}BN$	Peroxyisobutyrylnitrate
PMN	Polymorphonuclear leukocytes
PNH	Paroxysmal nocturnal hemoglobinuria
P_{O_2}	Oxygen tension
PP	Photoperiod
ppb	Parts per billion
ppb C	Parts per billion expressed as carbon
pphm	Parts per hundred million
ppm	Parts per million
ppm C	Parts per million expressed as carbon
PPN	Peroxypropionylnitrate
ppt	Parts per trillion
psi	Pounds per square inch
PST	Pacific standard time
Pst TLC	Maximal transpulmonary pressure
r	Correlation coefficient
R	Roentgen
RAPS	(St. Louis) Regional Air Pollution Study
Raw	Airway resistance

RBC	Red blood cells; erythrocytes
RCHO	Higher aldehydes
$RC(O)O_2$	Peroxyacyl radical
$RCOO_2$	Organic peroxy compounds
$RCOO_2H$	Organic peracids
$RC(O)O_2NO_2$	Peroxynitrates
RDW	Root dry weight
RH	Relative humidity
RHC	Reactive hydrocarbons
RL	Pulmonary resistance
RO_2	Alkoperoxy radical
$RONO_2$	Organic nitrates
RO_2NO_2	Organic peroxynitrates
ROOH	Organic hydroperoxides
rpm	Revolutions per minute
Rt	Flow resistance
RTI	Research Triangle Institute
RTP	Research Triangle Park
RV	Residual volume
S	Sulfur
-S-	Sulfide linkage
SAI	Systems Applications, Inc. (Model)
SBR	Styrene-butadiene rubber
S.D.	Standard deviation
sec	Second
SEM	Scanning electron microscopy
S.F.	San Francisco
SGaw	Specific airway conductance
SGPT	Serum glutamic-pyruvic transaminase
SH	Sulfhydryl group
SMSA	Standard metropolitan statistical area
SO_2	Sulfur dioxide
SO_3	Sulfur trioxide
SO_x	Sulfur oxides
SOD	Superoxide dismutase
sp. or spp.	Species
SPM	Suspended particulate matter
^{90}Sr	Radioisotope of strontium
SRI	Stanford Research Institute
SRM	Standard reference material
-SS-	Disulfide linkage
SSET	Small-scale eddy transport
STA	Seasonal tropopause adjustment
Std	Desired air quality (standard)
t	Student's statistic
TDW	Top dry weight
TF	Tropopause folding
TGV	Thoracic gas volume
TLC	Total lung capacity
UKI	Unbuffered potassium iodide
UV	Ultraviolet
V_{25}	Maximum expiratory flow rate at 25% of VC
V_{50}	Maximum expiratory flow rate at 50% of VC
VC	Vital capacity

VMT	Vehicle miles traveled
V_{O_2}	Oxygen uptake
V_{O_2} max	Maximum oxygen uptake
v/v	Volume/volume
W	Watts
wk	Week
wt	Weight
XO, XO_2	Compounds in which X represents hydrogen or an organic radical (R or RO)
yr	Year
α	Level of statistical significance set by investigator
μg	Microgram
$\mu g/m^3$	Microgram per cubic meter
μm	Micrometer
μmol or $\mu mole$	Micromole
$>$	Greater than
$<$	Less than
$\sim$	Approximately

ABSTRACT

This document is an evaluation and assessment of scientific information relative to determination of health and welfare effects associated with exposure to various concentrations of ozone and other photochemical oxidants in ambient air. The document is not intended as a complete, detailed literature review. It does not cite every published article relating to oxidants in the environment and their effects. The literature through 1976-1977 has been reviewed thoroughly for information relative to criteria. An attempt has been made to identify the major discrepancies in our current knowledge, again relative to criteria.

Though the emphasis is on presentation of health and welfare effects data, other scientific data are presented and evaluated to provide a better understanding of the pollutants in the environment. To this end, separate chapters are included on the nature and atmospheric concentrations of photochemical oxidants, their sources and removal processes, oxidant precursors, the relationships between ambient oxidants and precursor emissions, and measurement methods for ozone, oxidants, and their precursors.

Specific areas addressed within a general area of health or welfare effects are as follows: Toxicological, clinical, and epidemiologic appraisals, and effects on vegetation, ecosystems, and materials.

1. SUMMARY AND CONCLUSIONS

INTRODUCTION

This document consolidates and assesses current knowledge regarding the origin of ozone and other photochemical oxidant pollutants and their effects on health, vegetation, certain ecosystems, and materials. This chapter summarizes the information contained in this document and includes conclusions that are believed to provide a reasonable basis for evaluating the effects on health or welfare that are produced by various concentrations of ozone and other photochemical oxidants. Although nitrogen dioxide is considered one of the photochemical oxidants, oxides of nitrogen are the subject of a separate report and are therefore discussed in this document only as they participate in the formation and reactions of other photochemical oxidants.

Hydrocarbons (HC) and other organics are important air pollutants almost entirely because they are precursors of other compounds formed in the atmospheric photochemical system, not because they produce any direct effects themselves. Toxic organics are considered only with respect to eye irritation.

The studies and data cited constitute the best available basis for specific standards aimed at protecting human health and the environment from photochemical oxidants in ambient air.

NATURE AND ATMOSPHERIC CONCENTRATIONS OF PHOTOCHEMICAL OXIDANTS

Photochemical oxidants are products of atmospheric reactions involving organic pollutants, nitrogen oxides (NO_x), oxygen, and sunlight. They consist mostly of ozone, NO_2, and peroxyacetylnitrate (PAN), with smaller amounts of other peroxyacylnitrates and other peroxy compounds, and they are formed along with other photochemical products such as aldehydes, nitrous acid, nitric acid, and formic acid. Photochemical oxidants originate mainly from volatile organic and NO_x emissions produced by human activities. Photochemical oxidant formation is a complex function of emissions and meterological patterns.

Peak concentrations of oxidant, expressed as ozone, are generally higher in urban and suburban areas than in rural areas, reaching levels in excess of 590 $\mu g/m^3$ (0.3 ppm). In rural areas, peak concentrations are lower but often exceed the 1-hr National Ambient Air Quality Standard of 160 $\mu g/m^3$ (0.08 ppm). However, dosages or average concentrations in rural areas are comparable to or even higher than those in urban areas. Because of pollutant transport, oxidant pollution is a regional rather than a local problem.

SOURCES AND SINKS OF OXIDANTS

All of the evidence presently available shows that in and around urban centers that have severe oxidant/ozone pollution, photochemical oxidant formed from anthropogenic organics and NO_x is the major contributor. Since *Air Quality Criteria for Photochemical Oxidants* was issued in 1970, the mechanisms of atmospheric oxidant/ozone formation have been studied intensively and are now understood in greater detail. Most noteworthy are recent findings pertaining to the roles of hydroxyl ($OH\cdot$) and hydroperoxy ($HO\cdot_2$) radicals. The reaction with $OH\cdot$ has been established to be a major hydrocarbon-consuming process, and $OH\cdot$ and $HO\cdot_2$ have been identified as having major roles in the atmospheric oxidation of NO to NO_2. Aldehydes and PAN have also been found to play important roles in the atmospheric reaction. For olefins and paraffins, at least, these reactions are now understood to the extent that the kinetics of photochemical hydrocarbon-NO_x reaction systems, as observed in the laboratory, can be described with reasonable accuracy. Additional research is needed to explain the atmospheric reactions of aromatic hydrocarbons and to clarify further the differences between laboratory and ambient atmospheric chemical systems.

The photochemical formation of oxidant/ozone is the result of two coupled processes: (1) a physical process involving dispersion and transport of precursors to oxidants (e.g., HC and

NO_x), and (2) the photochemical reaction process. Both processes are strongly influenced by meteorological factors such as dispersion, solar radiation, temperature, and humidity. Recent data on wind velocity and mixing height show that episodes of limited dispersion are most common in the Far West and in the Rocky Mountains region, least common over the Plains States, and of intermediate frequency east of the Mississippi. New measurements of solar radiation indicate that the distribution of light intensity among respective wavelength intervals is different from that previously reported, which results in higher photodissociation constants for NO_2 in the ambient atmosphere. Recent field and laboratory studies suggest that at temperatures below approximately 55° to 60°F, concentrations of photochemical ozone are unlikely to exceed the national 1-hr standard of 160 $\mu g/m^3$ (0.08 ppm).

For understanding the sources of photochemical oxidants or ozone (oxidant/ozone), perhaps the most important recent development is the identification of short- and intermediate-range and synoptic-scale transport of photochemical oxidant/ozone. Short-range (urban-scale) transport causes the highest ozone concentrations some distance downwind from the core area (region of highest emission) of an urban center. Intermediate-range (mesoscale) transport occurs as urban oxidant/ozone plumes extending as far as 100 miles or more downwind and is also involved in land-sea breeze circulation. Finally, synoptic-scale transport over several hundred miles, associated with high-pressure systems, has been found to occur extensively. These findings have significant implications with respect to the location of oxidant/ozone monitoring stations.

Conditions during long-range transport are such that ozone production per unit of precursor is enhanced. Also, many organics previously thought to be unreactive are now believed to have significant ozone-producing potential.

In addition to photochemical reactions of anthropogenic emissions, potential sources of oxidant/ozone in the troposphere are the intrusion of stratospheric ozone and the photochemical reactions of natural organic and NO_x emissions. Estimates of ground-level concentrations of ozone originating in the stratosphere are based on two types of evidence: (1) global circulation patterns, namely, patterns in air interchange between stratosphere and troposphere; and (2) data on variations of ozone concentrations in remote rural areas. Based on the evidence of stratosphere-troposphere interchange, the annual average stratospheric contribution to ozone concentrations at ground level is estimated to be 43 to 98 $\mu g/m^3$ (0.022 to 0.05 ppm). The highest concentrations, at or above 160 $\mu g/m^3$ (0.08 ppm), from that source are expected to occur mainly during April and May. Analysis of ozone data for rural areas indicated that major intrusions of stratospheric ozone also occur during the spring months in mid-latitudes. The highest 1-hr concentration of stratospheric ozone reaching ground level during the smog season (usually late summer or early fall) has been found to range from 29 to 78 $\mu g/m^3$ (0.015 to 0.040 ppm), depending on the investigator. More recent data obtained at Whiteface Mountain, New York, suggest a maximum 24-hr concentration of 72 $\mu g/m^3$ (0.037 ppm) stratospheric ozone in July.

Certain organic emissions from vegetation (terpenes) play the dual role of oxidant/ozone precursor and scavenger. Despite the substantial rates at which they are emitted in forested areas, the ambient concentrations of such organics, because of their reactivity and the areal dispersion of their sources, seldom exceed a few parts per billion (ppb). At these concentrations, the *direct* potential of terpenes for photochemical ozone formation is estimated to be negligible. It is conceivable, however, that the products of atmospheric reactions involving large amounts of terpenes do have a significant impact on oxidant/ozone-related air quality.

OXIDANT PRECURSORS

Organic pollutants in urban atmospheres consist mainly of hydrocarbons from automobile exhaust and fuel evaporation, and of oxygenated hydrocarbons and halocarbons emitted mainly from manufacturing and from the use of organic chemicals. In urban atmospheres, total organic concentrations, reported usually as 6- to 9-a.m. averages of total nonmethane hydrocarbons (NMHC), are typically in the 1-ppm range and can be as high as 10 ppm or even higher, as in Los Angeles. In rural and remote atmospheres, the composition and concentration of organic pollutants are uncertain, largely because of deficiencies in the analytical methods available for determining the concentrations involved. Evidence suggests that NMHC levels are generally less than 0.1 ppm, a fraction of which consists of vegetation-related terpenes.

Concentrations of NO_x in urban atmospheres vary within a wide range, with highest values exceeding 1 ppm. Such concentrations appear to decrease rapidly as the air mass moves away from the city. In rural and remote areas, ambient concentrations do not exceed a few ppb and are often below the 5 ppb detection limit of current commercial NO_x analyzers.

Hydrocarbons and NO_x are emitted to the atmosphere from both natural and manmade sources, with natural sources probably contributing more on a global scale. Natural and anthropogenic sources are, however, generally segregated geographically, so that anthropogenic emissions are concentrated in the populated urban areas. Thus it is the latter that are most relevant to oxidant/ozone pollution problems downwind from populous areas. At present, mobile sources appear to account for the major part of the organic compounds emitted in most urban areas. The imposition of emission standards for mobile sources has reduced the reactive hydrocarbon component of emissions from gasoline-powered vehicles.

RELATIONSHIPS BETWEEN AMBIENT OXIDANT AND PRECURSOR EMISSIONS

Quantitative relationships between ambient oxidant/ozone and precursor emissions are needed for predicting the impact of these emissions on air quality. Such relationships represent, with varying degrees of complexity, the physical and chemical processes taking place in the atmosphere.

Air quality simulation models (AQSM's), a number of which are available, represent an approach to relating precursor emissions to oxidant air quality. However, AQSM's require a great deal of computation and have not been adequately evaluated.

In summary, satisfactory methods of relating the emission of precursors to oxidant concentrations in ambient air are not yet available. For this reason, the U.S. Environmental Protection Agency has been sustaining an intensive research effort to develop and validate satisfactory models that relate emissions to air quality. Based on results of this research and on model performance standards yet to be defined, the Agency will recommend specific models for use in formulating optimum ozone control strategies.

MEASUREMENT METHODS FOR OXIDANT AND OXIDANT PRECURSORS

The chemiluminescence method, based on the gas-phase reaction of ozone with ethylene, was adopted by EPA as the reference method and is now being used extensively. Several commercial instruments based on reference methods specified in EPA's *Ambient Air Monitoring Reference and Equivalent Methods* have been found to perform better than required by these EPA regulations. Techniques for continuous measurement, based on gas-solid chemiluminescence and ultraviolet (UV) photometry, have been developed and designated as equivalent methods.

Extensive studies in recent years have shown that various traditional KI calibration procedures for oxidant or ozone analyzers are deficient, and that these methods suffer from lack of both precision and accuracy.

Methods for measuring total oxidants are based on the oxidation of potassium iodide (KI) and the electrochemical or colorimetric detection of iodine (I_2). Nitrogen dioxide, however, causes a positive interference, and SO_2 causes a negative interference in these methods; these interferences are very difficult to correct for. EPA intends to study and evaluate several new calibration procedures and to amend Appendix D of 40 CFR Part 50 to revise the 1 percent neutral buffered potassium iodide (NBKI) calibration procedure or to replace it with one or more of several alternatives under consideration. These alternatives are: (1) gas-phase titration (GPT) with excess NO; (2) UV photometry; (3) GPT with excess O_3; and (4) boric acid KI.

Commercial instruments for monitoring total nonmethane hydrocarbon (NMHC) in ambient air have been tested extensively and found to be inaccurate at NMHC levels approaching the 0.24-ppm C air quality standard. At present, hydrocarbons in ambient air can be accurately measured only by sophisticated gas chromatography. Routine methods are under development.

On December 1, 1976, EPA promulgated a new principle and calibration procedure for measuring NO_2. The measurement principle is the gas-phase chemiluminescent reaction of O_3 and NO. Two calibration procedures are prescribed. One is a GPT procedure referenced to an NO-in-nitrogen standard. The other calibration procedure is referenced to an NO_2 permeation device. Before the development of chemiluminescence analyses, most NO_2 data were collected using methods based on variations of the Griess-Saltzman procedure. For all practical purposes, these methods have been replaced by the chemiluminescence method.

HEALTH EFFECTS OF OZONE AND OTHER PHOTOCHEMICAL OXIDANTS

In this section, the influence of exposure to ozone and other photochemical oxidants on physiology and health is assessed, beginning with a brief discussion of the concept of "threshold pollutant concentrations" and its application to the protection of public health. Then, the strength of association between exposure to ozone and other oxidants and changes in several types of biomedical indicators is evaluated.

For each type of indicator, the discussion addresses four main topics: (1) the degree to which changes in each type constitute impairments in public health; (2) the available scientific evidence relating ozone and other oxidant exposures to changes within each type; (3) the reliability of existing scientific evidence; and (4) where appropriate, the confidence with which findings may be attributed to ozone alone, as opposed to other substances or combinations of substances.

Whenever possible, clinical (human experimental) and epidemiologic studies will be discussed together. However, because of the great uncertainty inherent in the quantitative extrapolation of results of animal studies to humans, human and animal studies will be discussed separately.

Discussion of Threshold Concentrations

The Clean Air Act directs that National Primary Ambient Air Quality Standards be such that their attainment and maintenance shall, in the judgment of the Administrator, and allowing an adequate margin of safety, be requisite to protect the public health. The confidence with which a margin of safety can be defined depends on the precision with which a threshold pollutant concentration, (the level above which exposure to ozone or other oxidants promotes impairment of health and below which it does not) can be determined.

In practice, no single overall threshold concentration for ozone or other pollutants exists. Thresholds have been shown to vary widely with the population segment studied and the biologic indicators measured. Also, since the great majority of known dose-response relationships do not show sharp discontinuities, it is most unlikely that a discrete threshold pollutant concentration can be established even for a single population segment and a single biomedical indicator.

Despite these limitations, the environmental decisionmaker may find it useful to incorporate the concept of threshold concentrations into the standard-setting process. For a given population segment, a threshold concentration may be operationally defined as occurring somewhere between a concentration at which no effect on health or function has been observed, and a concentration at which such an effect has been demonstrated. In protecting public health, the population segments of primary concern are the most susceptible groups, in whom exposure to ozone or other pollutants is most likely to promote impairment of health. Such groups may include those with underlying illness, the very old, the very young, and the pregnant. (It has not been determined whether such susceptible population segments differ from the most sensitive population segments, which comprise those individuals most likely to show measurable responses to very low pollution concentrations.)

Ideally, the environmental decisionmaker would know the pollutant concentrations with which no adverse effects are associated in susceptible groups, as well as the concentrations with which such adverse effects are unambiguously associated. Unfortunately, knowledge in both of these areas remains sparse. Recent experimental studies of healthy people and animals have greatly advanced our knowledge of the health effects of ozone. However, threshold concentrations deducible from such studies may not apply to the potentially susceptible groups described above. The opportunity to study such groups experimentally is severely limited by practical and ethical constraints. The relatively few epidemiologic studies of these groups that are extant have yielded inconclusive results.

Human Studies

SHORT-TERM OXIDANT EXPOSURES

Mechanical Function Of The Lung

Assessment Of Health Effect. There is considerable room for honest disagreement on whether pollution-induced alterations in mechanical lung function constitute bona fide impairments of health in and of themselves. In the great majority of experimental studies in which oxidant exposures have produced changes in lung function in healthy subjects, function has returned to normal within a few hours. Thus there is no reason to suspect that in healthy individuals such changes promote any measurable increase in risk of future illness. Nor does any available evidence suggest that in healthy individuals a small change in ventilatory function, unaccompanied by symptoms or impairment of oxygen uptake or work capacity, would interfere with normal activity or task performance.

However, three considerations suggest that oxidant-associated changes in lung function may signal impairment of public health. First, in persons with underlying respiratory illness such as asthma, chronic bronchitis, and emphysema, even small decrements in lung function often interfere with normal activity. Second, at experimental ozone concentrations as low as 0.30 ppm, decrements in lung function have usually been accompanied by physical discomfort, as manifested in symptoms such as sore throat, chest pain, cough, and headache. At times this discomfort has been great enough to prevent the completion of experimental protocols, particularly when subjects have been exercising vigorously. It appears quite likely that the pulmonary irritant properties of ozone (and perhaps other oxidants) underlie both the discomfort and the decrements in function. Thus, at least when associated with ozone exposure, changes in lung function often represent a level of discomfort that, even among healthy people, may restrict normal activity or impair the performance of tasks.

Summary Of Data. Human experimental studies have demonstrated that the subject's level of exercise during ozone exposure is directly related to the magnitude of change in lung function and the severity of symptoms at any given ozone concentration. During exercise, subjects increase their expiratory flow rates, and they tend to breathe through their mouths. These factors increase the total dose of ozone delivered to the lung and may increase the depth to which it is delivered in the respiratory tree.

After 2 hr of resting exposure to 1470 $\mu g/m^3$ (0.75 ppm), healthy young adults showed small changes in lung function in a study by Bates et al. Folinsbee et al. observed changes in respiratory pattern (increased respiratory frequency and decreased tidal volume) and reductions of vital capacity in healthy young adults exercising submaximally after a similar resting exposure. Immediately after 2 hr of exposure to 1470 $\mu g/m^3$ (0.75 ppm) ozone, during which they performed intermittent light exercise, subjects showed quite pronounced changes in lung function in studies by Folinsbee et al. and Hazucha et al. In subjects exposed under the same conditions, Folinsbee et al. observed changes in respiratory pattern, though not in minute volume or oxygen uptake.

In subjects exercising maximally after 2 hr of exposure to 1470 $\mu g/m^3$ (0.75 ppm) ozone and intermittent light exercise, Folinsbee et al. observed decrements in maximum work load, tidal volume, heart rate, and oxygen uptake.

Folinsbee et al. observed decreases in tidal volume and maximum expiratory flow rate at 50 percent of vital capacity ($\dot{V}_{50}$) in subjects exercising submaximally after 2 hr of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise. In subjects giving no history of cough, chest discomfort, or wheezing in response to allergy or air pollution exposure (unreactive subjects), few changes in lung function occurred after 4 hr of exposure to 980 $\mu g/m^3$ (0.50 ppm) and intermittent light exercise (Hackney et al.). However, in subjects giving such a history (reactive subjects) and receiving an identical exposure, the same investigators observed decrements in 8 of 14 measured parameters. Interestingly, in a second group of unreactive subjects, the same investigators observed substantial decrements in lung function after only 2 hr of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise.

Statistically significant changes in forced vital capacity, maximum mid-expiratory flow rate, and airway resistance in 22 young males after 2 hr of exposure to 0.4 ppm ozone and intermittent moderate exercise were observed by Knelson et al. After subjects had been exposed for 4 hr, changes in these parameters had increased, and several other flow parameters had also changed significantly in comparison with control values.

In studies by Hazucha and Bates, Hazucha et al., and Folinsbee et al., subjects showed changes in lung function after 2 hr of exposure to 730 $\mu g/m^3$ (0.37 ppm) ozone and intermittent light exercise. Hackney et al. observed such changes in reactive subjects but not in unreactive subjects. In a separate study by Hackney and colleagues, four Canadians and four southern Californians were exposed in the Los Angeles area to 730 $\mu g/m^3$ (0.37 ppm) ozone for 2 hr. The Californians showed few changes in lung function, whereas the Canadians showed decrements in most parameters measured. (At least partly because of the small sample sizes, no observed changes were statistically significant.) In subjects exposed to 730 $\mu g/m^3$ (0.37 ppm) ozone together with 1000 $\mu g/m^3$

(0.37 ppm) SO_2, Hazucha and Bates observed an effect on lung function substantially larger than the sum of the separate effects of the individual pollutants.

DeLucia and Adams observed changes in lung function and respiratory pattern in healthy subjects exercising steadily and fairly heavily over a 1-hr exposure to 590 $\mu g/m^3$ (0.30 ppm) ozone. Two of six subjects experienced such discomfort as to prevent them from completing the experimental protocol.

Hazucha observed small changes in lung function in three nonsmokers exposed for 2 hr to 490 $\mu g/m^3$ (0.25 ppm) ozone and intermittent light exercise. No lung function changes of note were seen by Hackney et al. even among reactive subjects who were similarly exposed.

After 1 hr of exposure to 290 $\mu g/m^3$ (0.15 ppm) ozone and steady, fairly heavy exercise, subjects observed by DeLucia and Adams showed changes in respiratory pattern. In two of six subjects, the same investigators noted inconsistent increases in residual volume.

Small but statistically significant increases in airway resistance, as measured by plethysmography, were observed in two of four healthy subjects immediately after a 1-hr exposure to 200 $\mu g/m^3$ (0.1 ppm) ozone. The investigators (Goldsmith and Nadel) did not state whether the subjects exercised during exposure.

Von Nieding and Wagner reported that subjects showed decrements in arterial oxygen pressure (Pa_{o2}) and airway resistance after 2 hr of exposure to 200 $\mu g/m^3$ (0.10 ppm) ozone and intermittent light exercise.

Kagawa and Toyama reported the results of Japanese epidemiologic studies relating lung function in Japanese elementary school children to daily air pollution level. In approximately 25 percent of the children studied, lung function parameters were significantly correlated with the average ozone concentration in the 2 hr before testing. During these studies, the ozone concentration ranged between 20 and 590 $\mu g/m^3$ (0.01 and 0.30 ppm). Correlations of lung function with ozone exposure were generally greater than with total oxidant exposure, and correlations of ozone exposure with parameters reflecting upper airway function were generally greater than with those reflecting lower airway function. The design of these studies precludes the inference of specific dose-response relationships.

Reliability Of Evidence. Available evidence showing that 730 $\mu g/m^3$ (0.37 ppm) ozone has an effect on the lung function of lightly exercising subjects is convincing. Though it remains unreplicated, the study of DeLucia and Adams raises the distinct possibility that an ozone concentration of 590 $\mu g/m^3$ (0.30 ppm) exerts a temporary effect on the lung function of healthy subjects exercising fairly strenuously. The same investigators have raised the question as to whether ozone concentrations as low as 290 $\mu g/m^3$ (0.15 ppm) exert effects in a portion of healthy subjects exercising strenuously.

The findings of Nadel and Goldsmith suggest that certain changes in lung function may be detectable in some healthy subjects who have been exposed to 200 $\mu g/m^3$ (0.1 ppm) of ozone. For three reasons, however, these findings do not suggest any endangerment of public health. First, the subjects experienced no physical discomfort while exposed to 200 $\mu g/m^3$ (0.1 ppm) ozone. Second, the observed changes in airway resistance were small. Third, the two subjects who showed significant increases in airway resistance after exposure to 200 $\mu g/m^3$ (0.1 ppm) ozone showed smaller, nonsignificant increases in airway resistance after exposure to 780 $\mu g/m^3$ (0.4 ppm) ozone.

The studies of Von Nieding and Wagner, though interesting, are unconfirmed. These investigators did not use standard techniques to measure airway resistance; nor did they draw arterial blood for Pa_{o2} measurements. Instead, they drew arterialized blood from the subjects' ear lobes. Thus, until confirmed with generally accepted methods, these studies must be interpreted with caution.

The studies of Kagawa and Toyama, like those of DeLucia and Adams and of Hackney, suggest that even among healthy individuals there is a considerable range of sensitivity to ozone exposure. Whether the lung function of respiratory disease patients is more sensitive to such exposure than that of healthy persons has not been determined. However, as mentioned above, the clinical significance of changes in lung function is likely to be greater in respiratory disease patients than in healthy subjects.

The experimental study of Canadians and Californians by Hackney et al. is consistent with the hypothesis that repeated oxidant exposures promote adaptation toward the maintenance of full lung function. More study is required to confirm this hypothesis and to investigate whether adaptation in lung function is of any long-term consequence to health.

Attributability Of Effects To Ozone. Experimental studies have shown that ozone at concentrations observed in the ambient air can produce changes in mechanical lung function. The epidemiologic studies of Kagawa and Toyama, in which lung function was correlated more strongly with ozone exposure than with total oxidant exposure, are consistent with this finding. However, the degree to which epidemiologic observations may be attributed specifically to ozone remains in doubt, since Kagawa and Toyama were not able fully to separate the effects of ozone from the effects of other environmental factors.

Finally, the work of Hazucha and Bates suggests that the effect on lung function of ozone at 730 $\mu g/m^3$ (0.37 ppm) may be enhanced by an identical concentration of sulfur dioxide. Observation of this enhancement argues in favor of providing a margin of safety in a primary National Ambient Air Quality Standard for ozone. However, this observation does not support a quantitative recommendation for a safety margin, since it has not yet been determined whether such enhancement occurs at lower ozone concentrations or with substances other than sulfur dioxide.

Impairment Of Physical Performance — Decrements in physical performance are deterrents to personal satisfaction. In this respect, such decrements constitute impairment of public health. The degree to which oxidant exposures may promote decrements in physical performance has not been determined. However, Wayne et al. assessed the association between hourly oxidant concentrations and the proportion of a high school cross-country team failing to improve running times between successive track meets in southern California. Over six cross-country seasons, the correlation of average oxidant concentration in the hour before the race with the proportion of runners failing to improve times was 0.88. The corresponding correlation for both the first and second three-season periods was 0.945. A correlation this high denotes a very close numerical relationship between two variables.

During the period studied, hourly oxidant concentrations ranged from approximately 60 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm). Inspection of the data of Wayne et al. reveals no obvious relationship between unimproved running time and oxidant concentrations below 200 to 290 $\mu g/m^3$ (0.10 to 0.15 ppm), in spite of the high overall correlations mentioned above. Also, since the authors did not consider ozone separately from other oxidants, the specific contribution of ozone to the observed results cannot be determined from this study.

As far as can be ascertained, no replication of the study of Wayne et al. has appeared in the published literature. For three reasons, however, the data from this study are more trustworthy than most results of a single epidemiologic study. First, the correlations between hourly oxidant concentration and unimproved running time were unusually high. Second, as mentioned in Chapter 10 of this document, Herman, at the University of North Carolina, has analyzed the data of Wayne et al. as well as data from two additional seasons, and he has observed results similar to those of Wayne et al. Third, the results of Wayne et al. are qualitatively consistent with the results of the experimental lung function studies mentioned above, especially those of Folinsbee et al. In view of these experimental studies, it would also appear plausible that ozone contributed significantly to the results of Wayne et al.

Oxidant Effects In Asthmatics - In the United States, there are an estimated 6 to 8 million asthmatics, about 70 percent of whom are estimated to live in urban areas. Thus, the number of asthmatics who may be exposed to elevated oxidant concentrations is substantial.

Available epidemiologic evidence on the relationship between oxidant exposure and exacerbation of asthma is very limited. In 1961, Schoettlin and Landau reported that the proportion of selected asthmatics in the Pasadena area having attacks was significantly greater ($p < 0.05$) on days when the maximum hourly oxidant concentration exceeded 490 $\mu g/m^3$ (0.25 ppm) than on days when the corresponding concentration was below this level. However, the proportion of asthmatics having attacks on days of maximum hourly oxidant concentration above 250 $\mu g/m^3$ (0.13 ppm) was not significantly different from the corresponding proportion when the maximum hourly concentration was below this level. The authors did not state the actual percentage of asthmatics having attacks on days in any exposure category, though they did state that asthma attacks tended to coincide with elevated oxidant levels in 8 (6 percent) of 137 patients studied.

Because it does not present asthma attack rates, the Schoettlin and Landau report gives no indication whether increases in attack rates might have been expected at maximum hourly oxidant concentrations below 490 μg/m^3 (0.25 ppm). Nor does it allow any judgments as to the extent to which increased attack rates might be attributable specifically to ozone. Despite the considerable attention it has received, this report should be considered the preliminary investigation its authors intended it to be.

Respiratory Symptoms and Headache

Assessment Of Health Effect. As mentioned above, increased rates of respiratory symptoms and headache constitute impairment of public health. Even when mild, such symptoms are annoying. Even when reversible, they may restrict normal activity or limit the performance of tasks.

Summary Of Data. In nearly all experimental studies in which ozone exposures have been sufficient to produce changes in lung function, most subjects have reported respiratory symptoms. The most common symptoms have been throat tickle, substernal tightness, pain on deep inspiration, and cough. Wheezing, dyspnea, and headache have occurred less commonly. Symptom severity has increased with ozone concentration and exercise. In studies of heavy exercise, symptoms have occasionally been severe enough to prevent subjects from completing experimental protocols.

In an epidemiologic study in southern California, Hammer et al. assessed the association between daily maximum hourly oxidant concentration and rates of chest discomfort, cough, and headache among student nurses. Rates of each of these symptoms, whether unadjusted or adjusted for fever, began to increase in the following oxidant concentration ranges: chest discomfort, 490 to 570 μg/m^3 (0.25 to 0.29 ppm); cough, 590 to 760 μg/m^3 (0.30 to 0.39 ppm); and headache, 290 to 370 μg/m^3 (0.15 to 0.19 ppm). Adjusted and unadjusted rates of headache, however, were not unequivocally elevated below maximum hourly oxidant concentrations of 590 to 760 μg/m^3 (0.30 to 0.39 ppm).

Several Japanese investigators have assessed the association between daily pollutant concentrations and symptom rates among students. In one Japanese study, rates of sore throat, dyspnea, and headache were somewhat higher during the summer months on days when the oxidant concentration exceeded 200 μg/m^3 (0.10 ppm) than on days when it did not. Over a 1-year period, rates of respiratory symptoms and headache were higher on days when the oxidant concentration exceeded 290 μg/m^3 (0.15 ppm) than on days when it was lower than 200 μg/m^3 (0.10 ppm). Though there is reason to believe that the stated oxidant concentrations were daily maximum hourly averages, averaging times were not clearly presented in the report of this study.

Reliability Of Evidence. Because of their close correlation with the results of experimental studies, the results of Hammer et al. appear to be quite reliable, even though unconfirmed in this country. Their reliability is enhanced by the large number of person-days of observation (about 53,000) encompassed by the study. Though ozone levels were not considered in the Hammer et al. study, it is reasonable to hypothesize, in view of experimental studies, that ozone contributed substantially to observed increases in rates of cough, chest discomfort, and headache.

As nearly as can be determined from translations of original articles, the Japanese epidemiologic studies cited in this document were appropriately designed. However, it is very difficult to interpret their results. Also, at least at present, the applicability of these results to any oxidant pollution problem in the United States must be considered very limited.

In data analyses, Japanese investigators (like many U.S. investigators) have not been able fully to separate the effects of individual pollutants. It is conceivable that combinations of pollutants unique to Japan were necessary to promote the increased symptom rates observed there. Averaging times for pollutant measurements were not clearly stated in the Japanese studies. Therefore, it is often impossible to draw specific inferences as to dose and response. In addition, the degree to which differences in Japanese and U.S. cultural responses to air pollution may differentially affect symptom perception in the two countries has not been determined.

A problem more fundamental than any of these specific reservations, however, is that Japanese and U.S. investigators as yet have great difficulty in exchanging specific scientific concepts. Until the quality of scientific communication between these groups of investigators increases, the ability to interpret the Japanese studies and to apply them to situations in the United States will remain severely limited.

Oxidants And Eye Irritation - In that it is annoying and uncomfortable, the reversible eye irritation produced by exposure to ambient photochemical oxidants may legitimately be considered a marginal impairment of public health. However, whether such eye irritation is sufficient to impair performance or restrict normal activity has not been determined. Nor has any association between oxidant-mediated eye irritation and chronic eye disease been observed.

In epidemiologic studies, no symptom has been more consistently linked to oxidant exposure than eye irritation. In most studies reported before the publication in 1970 of *Air Quality Criteria for Photochemical Oxidants,* rates of eye irritation were observed to increase fairly steadily when oxidant concentrations ranged from 200 to 880 μg/m^3 (0.10 to 0.45 ppm). Hammer et al. found that rates of eye discomfort began to increase at oxidant concentrations of 290 to 370 μg/m^3 (0.15 to 0.19 ppm).

Evidence linking ambient photochemial oxidant exposures to eye irritation is convincing. However, the specific etiologic agent or agents remain unknown. Experimental studies have shown quite conclusively that ozone at ambient concentrations is not an eye irritant.

Oxidants and Mortality - Review of existing studies shows no consistent association between daily oxidant concentrations and daily mortality rates. As far as can be ascertained, no studies of oxidant exposures and mortality have been performed since the publication in 1970 of *Air Quality Criteria for Photochemical Oxidants.*

Other Effects Of Short-Term Ozone And Oxidant Exposure

Changes In Erythrocytes. In an experimental study, Buckley et al. observed increased rates of erythrocyte lysis in H_2O_2 after a 2¾-hr exposure of healthy subjects to 980 μg/m^3 (0.5 ppm) ozone and intermittent light exercise. These investigators also noted changes in the activity of several erythrocytic enzymes. Hackney et al. observed an increased in vitro lysis rate in erythrocytes of Canadians, but not in that of Californians following a 2-hr exposure of subjects to 730 μg/m^3 (0.37 ppm) ozone and intermittent light exercise.

Chromosomal Aberrations. Merz et al. reported chromosomal abnormalities in the lymphocytes of six subjects after they were exposed to 980 μg/m^3 (0.5 ppm) ozone for 6 or 10 hrs. However, a study by McKenzie et al. showed no increased rate of leukocyte chromosomal aberration in the lymphocytes of 30 subjects exposed for 4 hr to 780 μg/m^3 (0.4 ppm) ozone. In the few epidemiologic studies of oxidant exposure and chromosome morphology performed to date, factors other than differences in oxidant exposure have confounded observed results to the extent that no inferences can be drawn.

Biochemical Parameters. Buckley et al. observed reduced glutathione reductase activity and increased vitamin E and lipid peroxidation in human serum following exposure of subjects to 980 μg/m^3 (0.5 ppm) ozone and intermittent light exercise for 2¾ hr. Following a 2-hr exposure to 730 μg/m^3 (0.37 ppm) ozone and intermittent light exercise, the Canadians and Californians studied by Hackney et al. showed increases in serum vitamin E levels; but only in the Canadians were these increases statistically significant.

After subjects exercised steadily and vigorously throughout a 1-hr exposure to 590 μg/m^3 (0.30 ppm) ozone, DeLucia and Adams observed no changes in the following biochemical blood parameters: hemoglobin level, nonprotein sulfhydryl level, erythrocyte glucose-6-phosphate dehydrogenase activity, and glutathione reductase activity.

Clinical Significance. The clinical significance of ozone-mediated changes observed in studies of blood is not yet known. As yet, an epidemiologic study of oxidant effects on anemic individuals has not been done. Whether or not oxidant exposures promote changes in leukocyte chromosome morphology, the significance of the changes themselves is unknown. Finally, changes in serum parameters of the magnitude observed in experimental ozone studies have not yet been linked to any clinical diseases.

LONG-TERM OXIDANT EXPOSURES

With the exception of the Hackney et al. study of Californians and Canadians, no experimental studies of humans have as yet assessed the effects of long-term oxidant exposures. The few available epidemiologic studies of such exposures have yielded inconclusive results. Mahoney has observed an association between broad patterns of oxidant distribution and annual respiratory disease mortality in the Los Angeles area. However, no convincing association between lung cancer mortality and oxidant exposure has been shown. In some studies, a limited association between the frequency of chronic obstructive lung disease and oxidant exposure has been observed. In other studies, however, no such association has been apparent. No relationship between long-term oxidant exposure and acute respiratory disease incidence or change in lung function has been observed, though neither of these areas has been extensively investigated.

Most available epidemiologic studies of long-term oxidant exposure are difficult to interpret. As usually acknowledged by the authors themselves, factors other than pollution exposure may often have influenced the results observed. Some studies have proved difficult to evaluate because areas in which health variables were compared did not show clear-cut differences in pollution exposure. Thus human studies cannot yet provide the environmental decisionmaker with concrete information as to the effects of long-term oxidant exposures on public health.

Animal Toxicology Studies

ANIMAL INFECTIVITY EXPERIMENTS

Increased susceptibility of animals to bacterial infection following ozone exposure at 196 μg/m^3 (0.1 ppm) is described by several investigators (Coffin et al., 1968; Ehrlich et al., 1976; Gardner et al., 1974; Coffin and Gardner, 1972; Miller et al., 1978). Others (Goldstein et al., 1971a, 1971b; Goldstein and Hoeprich, 1972; Goldstein et al., 1974), using bacterial infection, have developed indices of infection for measuring the effects of ozone on the lungs of rodents. The effective concentration of ozone at which susceptibility to infection is increased is lower when either other pollutants or stress is combined with ozone exposure. These findings have definite human health implications, although different exposure levels may be associated with such effects in humans. These reactions in mice represent effects on basic biological responses to infectious agents, and there is no reason to believe that the pollutant-induced alterations of basic defense mechanisms that occur in mice could not occur in humans who possess equivalent defense systems. The extrapolation of these data to man is not supported by direct epidemiologic evidence that susceptibility to infection increases in persons exposed to ozone and other photochemical materials. However, the biochemical and cellular alterations described below for animals suggest that multiple epithelial and biochemical targets are perturbed by ozone exposure. Ozone-induced irritation of the major bronchi in man does occur at ozone concentrations approximating 490 μg/m^3 (0.25 ppm). For tracheal ozone concentrations greater than 100 μg/m^3 (0.05 ppm), it is predicted that a smaller inspired concentration would be required in man than in rabbits and guinea pigs to result in a given respiratory bronchiolar dose (Miller et al., 1977). A 3-hr exposure to 490 μg/m^3 ozone (0.25 ppm) has been shown to be sufficiently high to injure rabbit alveolar macrophages. Thus, depending on the extent of nasopharyngeal removal of ozone in man, a given level may be expected to produce similar effects in man and certain animals.

MORPHOLOGICAL ABNORMALITIES OF ANIMAL RESPIRATORY SYSTEMS

A range of morphological effects is noted in association with experimental ozone exposures of 392 to 1960 μg/m^3 (0.2 to 1.0 ppm). These effects vary from the reversible replacement of Type 1 with Type 2 alveolar cells (e.g., 392 μg/m^3 or 0.2 ppm ozone for 3 hr/day over 7 days) to emphysematous changes and terminal bronchiolar and alveolar damage (e.g., 784 μg/m^3, or 0.4 ppm, ozone for 6 hr/day, 5 day/wk, for 10 months; and 1058 μg/m^3, or 0.54 ppm ozone continuous for 3 months). Occurrence of these effects after long-term exposure to low concentrations increases the expectation that repeated or chronic exposures may have the potential for inducing similar effects in humans.

BIOCHEMICAL EFFECTS

There is an impressive variety of biochemical alterations associated with ozone exposures over the range of 196 to 1960 μg/m^3 (0.1 to 1.0 ppm). Though effects caused by levels of 980 μg/m^3 (0.5 ppm) and greater have definite toxic potential, the biological significance of those changes detected in exposures to 196 to 392 μg/m^3 (0.1 to 0.2 ppm) are more difficult to assess. These biochemical changes are significant, however, in demonstrating that there are ozone-induced effects at cellular sites and in organ systems distant from the lung. Biological reactions to ozone include an increase in the activity of enzymes that detoxify oxidizing chemical species. Increased enzymatic activity, as well as increased oxygen consumption, has been shown in some instances to be prevented, reversed, or diminished by increased vitamin E levels (vitamin E is an antioxidant). Thus increased enzymatic activity may represent a reaction to potential toxicity rather than being a toxic response itself. Nevertheless, such reactions represent the organism's response to ozone. Such perturbations in biological systems may pose a health risk to the population, particularly for susceptible individuals.

GENETIC AND TERATOGENIC POTENTIAL

A report by Zelac (1971) on chromosomal abnormalities in peripheral leukocytes of intact hamsters which were exposed to 392 μg/m^3 (0.2 ppm) ozone for 5 hr was not confirmed by Gooch et al. (1976). Although confirmation of the results may suggest another category of ozone-induced effects, the preliminary evidence does not suggest that these effects occur at lower levels of exposure.

IMPLICATIONS OF ANIMAL TOXICOLOGY DATA

Note that the difference between concentrations of ozone that produce toxicological effects in animals (as well as symptomatic and lung function changes in humans) and ambient air levels of ozone is much smaller than for nearly any other atmospheric pollutant. There is an unusual clustering and convergence of various toxicological, experimental human, and epidemiologically observed human effects in association with ozone concentrations ranging from 392 to 1176 μg/m^3 (0.2 to 0.6 ppm).

Chronic effects of long-term ozone exposure in man cannot be quantitatively related to specific ozone concentrations of short (hourly or daily) averaging times because of the long period of disease induction and the varied exposures of individuals during the period of induction. Epidemiologic studies may establish relationships between long-term ozone exposure and the risk of human chronic disease, but toxicological studies must be relied on in quantifying the ozone levels that may induce chronic effects.

Ozone Versus Oxidant Health Effects

Two characteristics of ozone and oxidant exposures should be cited with reference to public health: (1) ozone itself is a primary cause of most of the health effects reported in toxicological and experimental human studies, and the evidence for attributing many health effects to this substance alone is very compelling; and (2) the complex of atmospheric photochemical substances is known to produce health effects, some of which (eye irritation, for example) are not attributable to pure ozone but may be caused by other photochemical substances in combination with ozone.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON VEGETATION AND CERTAIN MICROORGANISMS

Since injury to vegetation by oxidants was first identified in 1944 in the Los Angeles Basin, our understanding of oxidant effects and of the widespread nature of their occurrence has increased substantially. The major phytotoxic components of the photochemical oxidant complex are ozone and peroxyacetylnitrate (PAN), although some data suggest that other phytotoxicants are also present. The peroxyacylnitrates are the most phytotoxic of the known photochemical oxidants; but because ozone is ubiquitous and associated with widespread injury to vegetation, it is the most important phytotoxic component of the photochemical oxidant complex.

The effects of photochemical oxidants on vascular plants occur at several levels, ranging from the subcellular to the organismic, depending on the concentration and duration of exposure to the pollutant and the interval between cessation of exposure and examination of the plant.

The earliest effect is an increase in cell membrane permeability. Following that, cellular and biochemical changes take place that are ultimately expressed on the organismic level as visible foliar injury, increased leaf drop, reduced plant vigor, reduced plant growth, and death. Such biochemical modifications in an individual plant are manifested by changes in plant communities and, finally, in whole ecosystems.

Leaf stomata are the principal sites through which ozone and PAN enter plants. Oxidants affect photosynthesis, respiration, transpiration, stomatal opening, and metabolic pool development, as well as biochemical pathways and enzyme systems.

Visible injury is identifiable as pigmented, chlorotic, or necrotic foliar patterns. Metabolic cellular disturbances can occur without visible injury and may be reversible. However, most of the growth effects reported until recently were accompanied by visible injury.

Classic ozone injury is demonstrated by the upper-surface leaf fleck of tobacco and the leaf stipple of grape. Many plants show an upper-surface response with no associated injury to the lower surface of leaves. However, in monocotyledonous plants such as grasses or cereals, and in some non-monocotyledonous plants, there is no division of mesophyll tissue, and bifacial necrotic spotting (flecking) is a common symptom of ozone injury.

Coniferous trees exhibit different symptoms. Ozone is probably the cause of emergence tipburn in eastern white pine (white pine needle dieback) and of chlorotic decline, a needle injury of ponderosa pine.

Classic PAN injury appears as a glaze followed by bronzing of the lower leaf surface of many plants. Complete collapse of leaf tissue can occur if concentrations are sufficiently high. Early leaf senescence and abscission usually follow the chronic symptoms. Patterns of chronic injury are generally not characteristic and may be confused with symptoms caused by biotic diseases, insect infestation, nutritional disorders, or other environmental stresses.

A great deal of research has been done to define more accurately the effects of oxidants on plant growth and yield. Studies comparing the growth of plants in field chambers provided with carbon-filtered or nonfiltered ambient air containing oxidants have reported up to 50-percent decreases in yield of citrus (orange and lemon) exposed to oxidants; a 10- to 15-percent suppression in grape yield in the first year and a 50- to 60-percent reduction over the following 2 years; and a 5- to 29-percent decrease in yield of cotton lint and seed in California. Losses of 50 percent in some sensitive potato, tobacco, and soybean cultivars have been reported in the eastern United States. It is apparent that oxidants in the ambient air reduce the yields of many sensitive plant cultivars.

Experimental chambers with controlled environments have been used to study both short-term and long-term effects of exposure to ozone (Tables 11-2, 11-4, Chapter 11). Radishes given one, two, or three acute exposures (785 $\mu g/m^3$, or 0.40 ppm) of 1.5 hr each at 7, 14, and/or 21 days of age exhibited reductions in root growth. The reductions in root growth from the multiple ozone exposures were equal to the additive effects of the single exposures. In other words, the temporal distribution was not a significant factor. When soybean plants were exposed to 1468 $\mu g/m^3$ (0.75 ppm) ozone for 1 hr, root growth was consistently reduced more than top growth. There were also reductions in nodule weight and number. The greater reduction of root growth compared to top growth is related to the transport of photosynthate. Ozone also affects nitrogen fixation in clover, soybean, and pinto bean through reduction in nodule number, even though nodule size and efficiency of nitrogen fixation are not influenced. The effect of ozone on the number of nodules formed by legumes, if widespread, could have a major impact on plant communities and could affect fertilizer requirements. There are indications that the effect of ozone on nodulation may be related to the carbohydrate supply in the host plant.

Experimental long-term exposures to ozone of a variety of crops, as well as of ornamental and native plants, have resulted in a reduction in growth and/or yield. Exposure of 14 species representative of the aspen plant community to ambient air containing 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm) ozone and to ozone concentrations of 290 and 588 $\mu g/m^3$ (0.15 and 0.30 ppm) for 3 hr/day, 5 days/week, and to charcoal-filtered air throughout the growing season, resulted in foliar injury to all species at the highest pollutant concentration. The growth of two soybean cultivars (Hood and Dare) was inhibited by intermittent exposure to ozone at 196 $\mu g/m^3$ (0.10 ppm) for 3 weeks. Both root and top growth were decreased. Similar results were noted with radish, except that a lower concentration of ozone (98 $\mu g/m^3$, or 0.05 ppm) inhibited growth. In these studies, the reduced growth occurred even though there were few visible symptoms of plant injury.

A 30-percent reduction in the yield of wheat occurred when wheat at anthesis was exposed to ozone at 392 $\mu g/m^3$ (0.2 ppm) for 4 hr/day for 7 days. A significant reduction in the yield of tomato was noted when plants were experimentally treated with ozone at 686 $\mu g/m^3$ (0.35 ppm); fewer fruit set, and thus fewer fruit were harvested. Chronic exposures to ozone at 98 to 290 $\mu g/m^3$ (0.05 to 0.15 ppm) for 4 to 6 hr/day reduced yields in soybean and corn grown under field conditions. The threshold for measurable effects for ozone appears to be between 98 to 196 $\mu g/m^3$ (0.05 to 0.10 ppm) for sensitive plant cultivars. This is well within the range of ozone levels monitored in the eastern United States. Growth or flowering effects were reported for carnation, geranium, radish, and pinto bean grown in greenhouse chambers and exposed to ozone at 98 to 294 $\mu g/m^3$ (0.05 to 0.15 ppm) for 2 to 24 hr/day.

The two most critical factors in determining plant response to air pollution are duration of exposure and concentration of pollutants. These two factors describe exposure dose. In determining the response of vegetation to oxidants, concentration is more important than time.

The concept of limiting values was used by Jacobson to define a boundary between doses of a pollutant that are likely to injure vegetation measurably and those that are not. Foliar injury was used as the index of plant response. The ranges for limiting values for effects of ozone are:

1. Trees and shrubs -
   400 to 1,000 $\mu g/m^3$ (0.2 to 0.51 ppm)
   for 1 hr

  - 200 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 2 hr
  - 120 to 340 $\mu g/m^3$ (0.06 to 0.17 ppm) for 4 hr

2. Agricultural crops -
   - 400 to 800 $\mu g/m^3$ (0.2 to 0.41 ppm) for 0.5 hr
   - 196 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 1 hr
   - 75 to 180 $\mu g/m^3$ (0.04 to 0.09 ppm) for 4 hr

Limiting values for PAN are:
- 1000 $\mu g/m^3$ (0.2 ppm) for 0.5 hr
- 500 $\mu g/m^3$ (0.1 ppm) for 1 hr
- 175 $\mu g/m^3$ (0.035 ppm) for 4 hr

Doses of ozone or PAN greater than the upper limiting values are likely to cause foliar injury.

The data points used to determine the limiting values listed above are not necessarily threshold values, but are based on available published research data. More than 200 studies were surveyed. Any limitations that were present in the experimental techniques used in the studies are therefore expressed in the data points. The number of studies used to derive the data points for ozone exposure of trees and shrubs (19) and for PAN is another limitation on the values given above.

For agricultural crops, the inaccuracies in measurements make the interpretation of results of repeated long-duration exposures difficult. Thus limiting values for ozone concentrations below 100 $\mu g/m^3$ (0.05 ppm) are not useful.

An ozone concentration of 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm) for 4 to 6 hr/day for 15 to 133 days can significantly inhibit plant growth and yield of certain species (Table 11-4, Chapter 11).

Plant sensitivity to ozone and PAN is conditioned by many factors. Genetic diversity in sensitivity to ozone between species and between cultivars within a species is well documented. Variations in sensitivity to ozone within a natural species are well known for several pine species, including white, loblolly, and ponderosa. Plant sensitivity to oxidants can be changed by both climatic and edaphic factors. A change in environmental conditions can initiate a change in sensitivity at once, but it will be 3 to 5 days before the response of the plant is totally modified. Plants generally are more sensitive to ozone when grown under short photoperiods, medium light, medium temperature, high humidity, and high soil moisture. Injury from PAN may increase with an increase in light intensity. Conditions during exposure and growth affect the response of plants to oxidants in similar ways. In general, environmental conditions optimum for plant growth tend to increase the sensitivity to ozone. Factors that increase water stress at the time of exposure tend to make plants more tolerant to ozone. Soil moisture is probably the most important environmental factor that affects plant response to oxidants during the normal growing season. Physiologic age affects the response of the leaf to oxidants. Young leaf tissue is most sensitive to PAN, whereas newly expanding and maturing tissue is most sensitive to ozone. Light is required for plant tissue to respond to PAN; a similar light requirement is not needed for plants to respond to ozone.

The majority of effects observed, such as suppression of root growth, mineral uptake, and nitrogen fixation, apparently result from a suppression of photosynthesis and modifications in photosynthate distribution. This suppression of metabolic reserves ultimately slows plant growth and renders the plant more sensitive to other stresses. Physiological changes can provide a sensitive means of monitoring the health and vigor of the plant with or without visible injury. Ozone affects pollen germination in some species and may affect yield through incomplete pollination of flowers. Investigations with *Arabidopsis thaliana* showed no mutagenic effects from ozone over seven generations.

Mixtures of ozone and SO_2 can cause effects below the levels caused by either gas alone; however, there is some disagreement concerning the interactions of ozone with other gases. Ratios of gas mixtures, intermittent exposures, sequential exposures to pollutants, and predisposition by one pollutant to the effects of a second pollutant may be important factors in nature, but insufficient knowledge is available for elucidation of the effects.

The response of plants to oxidants may be conditioned by the presence or absence of biotic pathogens. Depending on the plant and the pathogen, oxidants may cause more or less injury to a given species. Oxidant injury to ponderosa pine predisposes the trees to later invasion by bark beetles. Ozone and ozone/sulfur dioxide mixtures can decrease the population of some plant-parasitic nematodes. Variable plant responses were noted when herbicides were used in the presence of high oxidant concentrations.

Little research on the effects of oxidants on ferns, nonvascular green plants, and microorganisms has been reported. Lichens and mosses are responsive to acid gases, but there is no definite evidence that they respond to oxidants. Ferns may be especially sensitive, but their injury response is different from that of higher vascular plants. Growth and sporulation of fungi on surfaces are usually, but not always, affected. Ozone from 0.1 to several milligrams per liter of solution is required to kill many microorganisms in liquid media. Most work with microorganisms has been done to study the effectiveness of ozone as a biocide in the storage of vegetation or in the treatment of water or sewage.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON ECOSYSTEMS

Plants, animals, and microorganisms usually do not live alone but exist as populations. Populations live together and interact as communities. Communities, because of the interactions of their populations and of the individuals that make them up, respond to pollutant stress differently from individuals. Man is an integral part of these communities, and as such, he is directly involved in the complex ecological interactions that occur within the communities and within the ecosystem of which the communities are a part.

The stresses placed on the ecosystems and their communities can be far-reaching, inasmuch as the changes that occur may be irreversible. For example, it has been suggested that the arid lands of India are the result of defoliation and elimination of vegetation, which in turn induced local climatic changes that were not conducive to the reestablishment of the original vegetation.

An ecosystem (e.g., the planet earth, a forest, a pond, or a fallen log) is a major ecological unit made up of living (biotic) and physical (abiotic) components through which the cycling of energy and nutrients occurs. A structured relationship exists among the various components. The biotic units are linked together by functional interdependence, and the abiotic units constitute all of the physical factors and chemical substances that interact with the biotic units. The processes occurring within the biotic and abiotic units and the interactions among them can be influenced by the environment.

Ecosystems tend to change with time. Adaptation, adjustment, and evolution are constantly taking place as the biotic component, the populations, and the communities of living organisms interact with the abiotic component in the environment. Recognizable sequential changes occur. With time, populations and communities may replace one another. This sequential change, termed succession, may culminate in climax communities. Climax communities are structurally complex, are more or less stable, and are held in a steady state through the operation of a particular combination of biotic and abiotic factors. The disturbance or destruction of a climax community or ecosystem results in its being returned to a simpler stage. Existing studies indicate that changes occurring within ecosystems, in response to pollution or other disturbances, follow definite patterns that are similar even in different ecosystems. It is therefore possible to predict broadly the basic biotic responses to the disturbance of an ecosystem.

Diversity and structure are most changed by pollution as a result of the elimination of sensitive species of flora and fauna and of the selective removal of the larger overstory plants in favor of plants of small stature. The result is a shift from the complex forest community toward the less complex hardy shrub and herb communities. The opening of the forest canopy changes the environmental stresses on the forest floor, causing differential survival and, consequently, changed gene frequencies in subcanopy species.

Associated with the reduction in diversity and structure is a shortening of food chains, a reduction in the total nutrient inventory, and a return to a simpler successional stage.

It should be emphasized that ecosystems are usually being subjected to a number of stresses at the same time, not just a single perturbation such as oxidant pollution.

The effects of oxidants on the mixed-conifer forest of the San Bernardino Mountains graphically demonstrate the changes that occur in natural ecosystems as discussed above. Since the early 1940's, the San Bernardino Forest has been undergoing stress from oxidants transported long-range from Los Angeles, 144 km (90 miles) away. Losses of ponderosa and Jeffrey pines, the overstory vegetation, have increased dramatically as pollutant levels have risen. Black oak has also suffered oxidant injury. The composition of both plant and animal populations has been altered by the death of the ponderosa and Jeffrey pines.

The interaction of pollutant and inversion layers at the heated mountain slope results in the vertical venting of oxidants over the mountain crest by upslope flow, thus establishing an elevational gradient of oxidant concentrations. Oxidant concentrations ranging from 100 to 200 $\mu g/m^3$ (0.05 to 0.10 ppm) at altitudes as high as 2432 m, approximately 1033 m above the mountain crest, have been measured by aircraft.

Total oxidant concentrations in the San Bernardino Mountains have been measured continuously from May through September since 1968 at the Rim Forest-Sky Forest monitoring station. During each of the first 7 years of monitoring, between June and September, the total number of hours in which concentrations of ozone were 160 $\mu g/m^3$ (0.08 ppm) or more was never less than 1300. The number of hours in which the total oxidant concentration was 390 $\mu g/m^3$ (0.20 ppm) or higher increased from fewer than 100 in 1969 to nearly 400 in 1974. It was not uncommon to observe momentary oxidant peaks as high as 1180 $\mu g/m^3$ (0.60 ppm). The duration of oxidant concentrations exceeding 200 $\mu g/m^3$ (0.10 ppm) was 9, 13, 9, and 8 hr/day going from the lower- to the higher-altitude stations.

The most recent data firmly indicate that oxidant concentrations in the San Bernardino Forest will either increase annually or oscillate around the mean of present high concentrations in the foreseeable future.

The transport of the urban plume from the coast northeastward to the mountains can be readily demonstrated. Because of this transport, the permanent vegetation constituting natural ecosystems receives much greater chronic exposure, while the short-lived vegetation constituting the economically more valuable agroecosystem of the Los Angeles coastal plain can be subject to injurious doses, but in intermittent, short-term fumigations. Each situation has measurable economic and aesthetic consequences, but on different time scales. The single-species agricultural ecologic system (the agroecosystem) has little resilience to pollutant stress. Losses are sometimes immediate and occasionally catastrophic. The complex natural ecosystem is initially more resistant to pollutant stress, but the longer chronic exposures cause disruption of both structure and function in the system that may be irreversible.

The oxidant injury to the mixed conifer stands of the San Bernardino Mountains that began in the early 1940's, as indicated above, is well advanced. A similar problem is developing in the forests of the southern Sierra Nevada Mountains. Both areas show direct as well as indirect effects on all subsystems of the forest ecosystem: producers, consumers, and decomposers.

In summary:

1. Ozone injury has limited the growth and caused the death of ponderosa and Jeffrey pines. An estimated 1.3 million trees have been affected. Decrease in cone production has resulted in a decrease in reproduction. Black oak has also suffered injury from ozone.
2. Reduction in fruits and seeds that make up the diet of most of the common small mammals has influenced the populations of these organisms.
3. Essential processes, such as recycling of nutrients, may have been disrupted, causing a limitation in the growth of vegetation.
4. Death of the predominant vegetation has caused an alteration in the species composition and a change in the wildlife habitat.

The San Bernardino Mountain study illustrates the complexity of the problems caused by environmental pollution. The changes that have occurred in this mountain ecosystem as the result of oxidant transport have already influenced the importance and value of this natural resource to the residents of Southern California.

The injury to the eastern white pine in the Appalachian Mountains caused by oxidant transport from the urban northeast has begun a similar sequential change that could degrade this important recreational area. Total oxidant peaks as high as 220 $\mu g/m^3$ (0.11 ppm) were recorded for July 1975. Concentrations exceeding 160 $\mu g/m^3$ (0.08 ppm) were measured in June 1976. These episodes resulted in significant increases in oxidant injury to three categories of eastern white pine in the Blue Ridge Mountains (the eastern range of the Appalachian Mountains).

Ecosystems are usually evaluated by modern man solely on the basis of their economic value. This economic value in turn depends on the extent to which man can manipulate the ecosystem for his own purposes. This single-purpose point of view makes it difficult to explain the many benefits of a natural ecosystem to man's welfare in terms of the conventional cost-benefit analysis. Gosselink, Odum, and Pope have, however, placed a value on a tidal marsh by assigning monetary values to its multiple contributions to man's welfare such as fish nurseries, food suppliers, and waste-treatment functions of the marsh. They estimate the total social value to range from $50,000 to $80,000 per acre.

Westman also evaluated the benefits of natural ecosystems by estimating the monetary costs associated with the loss of the free services (absorption of air pollution, regulation of global climate and radiation balance, and soil bioding) provided by the ecosystems. Westman estimated that the oxidant damage to the San Bernardino National Forest could result in a cost of $27 million per year (1973 dollars) just for removal of sediments resulting from erosion, as long as the forest remained in the early stages of succession.

Estimates of the cost in currency of the values of items and qualities such as clean air and water, untamed wildlife, and wilderness, once regarded as priceless, are an attempt to rationalize the activities of civilization. When estimating the monetary cost in currency of the values lost through damage to ecosystems, the assumption is usually made that the decisionmakers will choose the alternative that is most socially beneficial as indicated by costs compared to benefits. As Westman points out, the assumption that decisions maximizing benefit/cost ratios simultaneously optimize social equity and utility is based on certain inherent corollaries:

> (1) The human species has the exclusive right to use and manipulate nature for its own purposes. (2) Monetary units are socially acceptable as means to equate the value of natural resources destroyed and those developed. (3) The value of services lost during the interval before the replacement or substitution of the usurped resource has occurred is included in the cost of the damaged resource. (4) The amount of compensation in monetary units accurately reflects the full value of the loss to each loser in the transaction. (5) The value of the item to future generations has been judged and included in an accurate way in the total value. (6) The benefits of development accrue to the same sectors of society, and in the same proportions, as the sectors on whom the costs are levied, or acceptable compensation has been transferred. Each of these assumptions, and others not listed, can and have been challenged.

In the case of (4) above, for example, the losses incurred when developing natural ecosystems are involved affect species other than man. These losses are seldom, if ever, compensated. The public at large also is usually not consulted to determine whether the dollar compensation is adequate and acceptable. Frequently, there is no direct compensation. Corollary (5) can never be fulfilled because it is impossible to determine accurately the value to future generations.

Evaluating the contribution of functioning natural ecosystems to human welfare is a very complex task and involves weighing both economic and human social values. As life support systems, they should not be evaluated in economic terms.

With the passage of time, man has destroyed many of the naturally occurring ecosystems of which he was a part and has replaced them with simplified ecosystems wholly dependent on his care and protection and requiring a large input of energy.

Man favors the simple unstable and synthetic ecosystems because when extensively managed, and subsidized by the use of fossil fuels, they are highly productive. An agricultural ecosystem (agroecosystem) is an example of such a simplified ecosystem. The effects of oxidants on agroecosystems have been under study for more than 20 years. The study of effects on natural ecosystems is much more recent.

Plants grown in agroecosystems are largely annuals and can be replaced when they are susceptible to pollutant stress. Natural ecosystems remain in place year after year. Manmade pollutants are undoing relationships developed within these ecosystems over millions of years.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON MATERIALS

Ozone is a major factor in the overall deterioration of several different types of organic materials. In fact, certain specific organic compounds are more sensitive to ozone attack than are humans or animals. The magnitude of damage is difficult to assess because ozone is one of many oxidizing chemicals in the atmosphere that contributes to the weathering of materials. Nevertheless, researchers have shown that ozone accelerates the deterioration of several classes of materials, including elastomers (rubber), textile dyes and fibers, and certain types of paints and coatings.

Although many organic materials have been shown to be susceptible to ozone attack, only certain paints, elastomers, and dyes sustain damage representing significant economic loss. Even the measures to prevent ozone damage to elastomers and dyes constitute a major cost.

2. INTRODUCTION

Air quality criteria are expressions of the scientific knowledge of the relationships among various concentrations, averaged over an appropriate time period, of air pollutants in the atmosphere and their adverse effects on human health and the environment. Criteria are issued to assist in the formulation of decisions regarding the need for control of a pollutant and the development of air quality standards governing the pollutant. Air quality *criteria* are descriptive; that is, they describe the effects that have been observed when the ambient air concentration of a pollutant has reached or exceeded a specific figure for a specified period of time.

Many factors must be considered when developing criteria. Consideration must be given to the chemical and physical characteristics of the pollutants, the techniques available for measuring these characteristics, and the exposure time, relative humidity, and other environmental conditions. Natural and anthropogenic emissions must be considered and assessed. The number and distribution of sources and the extent to which the specific pollutant is emitted into the environment directly affect the levels to which the receptor is exposed. In addition, the criteria must also include consideration of the influence of all such variables on the effects of air pollution on human health, agriculture, vegetation, wildlife, visibility, and climate. Furthermore, the individual characteristics of the receptor must be taken into account.

Air quality *standards* are prescriptive. They prescribe pollutant exposures or levels of effect that scientific judgment determines should not be exceeded in a specified geographic area, and they are used as one of several factors in designing legally enforceable pollutant emission standards.

This document focuses on photochemical oxidants and their precursors, HC and NO_x, as they are found in the ambient air. The nitrogen oxides are examined, but only for their oxidant-precursor role; a detailed examination of NO_x as pollutants in their own right can be found in *Air Quality Criteria for Nitrogen Oxides* (U.S. Environmental Protection Agency, Publication No. AP-84, January 1971) and *Air Quality Criteria for Nitrogen Oxides* (Luxembourg: NATO/CCMS, Document N. 15, June 1973). The discussion of HC in this document rests almost entirely on their role as precursors of other compounds formed in the atmospheric reaction system and not on the direct effects of HC themselves. Gas-phase HC and certain of their oxidation products that are associated with the manifestations of photochemical air pollution are discussed. Toxic organics will not be discussed here. Establishing health data for toxic organics is extremely complex and will be handled at a later date as more studies are completed.

This publication reviews the sources of oxidants and of oxidant precursors and discusses the chemistry of the atmospheric oxidant formation process and the relationships between oxidants and their precursors. Air quality data, control techniques, and source and emission inventories are discussed briefly for orientation purposes only, inasmuch as these topics are discussed in detail in other documents. Measurement techniques and fate and transport of pollutants are discussed to the extent necessary to give a clear understanding of the health and welfare effects and of the bases for sound oxidant control strategies.

The status of control technology for oxidants and their precursors has not been treated. For information on this subject, the reader is referred to *Control Techniques for NO_x and Hydrocarbons from Mobile Sources* (U.S. Department of Health, Education, and Welfare, Publication No. AP-66, March 1977), and *Control Techniques for Hydrocarbons and Organic Solvents* (U.S. Department of Health, Education, and Welfare, Publication No. AP-68, March 1977). The subject of adequate margin of safety stipulated in Section 108 of the Clean Air Act also has not been treated. Again, the reader is referred to documentation prepared by OAQPS.

Methods and techniques for controlling the sources of photochemical oxidants as well as the costs of applying these techniques are discussed in other documents.

The scientific literature has been reviewed through 1976-77. This document is not intended as a complete, detailed literature review, and it does not cite every published article relating to oxidants in the environment and their effects. The literature has been reviewed thoroughly for information relative to criteria. Chapters 11 (Effects of Photochemical Oxidants on Vegetation and Certain Microorganisms) and 12 (Ecosystems) in this document are based on the chapters by the same numbers and titles in the National Academy of Sciences publication, *Ozone and Other Photochemical Oxidants* (Washington, D.C., 1977). An attempt has been made to identify the major deficiencies in our current knowledge relative to criteria. Between the issuance of the preprint volumes and the final publication of this document, a few minor changes were made to correct inadvertent omissions.

3. NATURE AND ATMOSPHERIC CONCENTRATIONS OF PHOTOCHEMICAL OXIDANTS

NATURE OF OXIDANT

Photochemical oxidants are chemical entities of concern because of their detrimental effects on biological systems and on certain materials. They are products formed in the atmosphere by sunlight-driven, chemical reactions that involve HC* and NO_x.

The term "photochemical oxidants" is used here to define those atmospheric pollutants that are photochemical reaction products and are capable of oxidizing neutral iodide ions. Extensive research has unequivocally identified several components of the photochemical oxidants mixture. Thus oxidants in ambient air are known to consist mainly of ozone, PAN, and nitrogen dioxide (NO_2) and are suspected to include also (but in smaller amounts) other peroxyacylnitrates, hydrogen peroxide, alkyl hydroperoxides, nitric and nitrous acids, peracids, and ozonides. Collectively, they are measured by potassium iodide procedures and are referred to in this report as "oxidant." An important distinction to be made here is that the formation of NO_2 in ambient air clearly precedes the formation of the other oxidants. For this reason, the relative levels of nitrogen dioxide and of the aggregate of the other oxidants vary considerably during the day, with NO_2 being invariably the dominant oxidant earlier in the day. Because of this difference in the variation pattern and the differences in effects between NO_2 and the aggregate of the other oxidants, the NO_2 pollution problem has been treated independently of the other photochemical oxidant problem. Furthermore, because of its predominance among oxidants other than NO_2, ozone has been singled out and treated as the sole representative of such oxidants and has been given most of the research attention.

Photochemical air pollution is customarily defined in terms of the concentrations of ozone and NO_2 only. This should not be interpreted to suggest that other photochemical pollutants are thought to be of less concern. Rather, it reflects (1) the fact that ozone and NO_2 pollution problems are more easily quantified and hence are more amenable to research than problems associated with the other pollutants (e.g., eye irritation and visibility reduction), and (2) the assumption that abatement of the ozone and NO_2 pollution problems will, in all probability, alleviate the other photochemical pollution problems.

The ozone found in the lower levels of the earth's atmosphere has been traced to both natural and anthropogenic sources. One natural source of tropospheric ozone is the ozone abundantly present in the stratosphere, which can be transported into the biosphere. Ozone can also form naturally from electrical discharges in the atmosphere and from atmospheric photochemical reactions involving naturally emitted organic vapors and NO_x. Obviously, the levels of ozone resulting from all such uncontrollable sources must be known if the anthropogenic sources and the effects of their control are to be assessed reliably.

Ozone and other oxidants from anthropogenic sources are products of atmospheric photochemical reactions involving primary organic and inorganic pollutants and atmospheric oxygen. More specifically, the oxidant formation process is initiated by the photolysis of light-absorbing air contaminants such as NO_2, aldehydes, and ozone, resulting in the formation of highly reactive radicals that react subsequently with organic pollutants. The net result of this photochemical activity is the accumulation of ozone, and other oxidants, to concentrations that depend on many factors, including local meteorological conditions (sunlight intensity, air stagnation, temperature, etc.) as well as the concentrations, makeup, and variation patterns of the primary pollutants present. Such dependence of oxidant formation on multiple factors makes this pollution problem immensely complex.

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*The term "hydrocarbon" is meant to include also all nonhydrocarbon organic compounds capable of participating in the atmospheric oxidant formation process. Accordingly, the terms "hydrocarbons" and "organics" are often used interchangeably.

OXIDANT CONCENTRATIONS AND THEIR PATTERNS

Introduction

Although buildup of photochemical oxidants occurs in nearly every urban center in the United States, no pollution episodes involving a sudden and massive assault on human health have been attributed solely to photochemical oxidants. Accordingly, no case studies have been reported in which pollutants and their effects were comprehensively and systematically examined during an oxidant pollution episode. Photochemical oxidants are viewed as an air pollution problem mainly on the bases of (1) observations of humans, animals, plants, and materials in areas known to have high oxidant levels, and (2) results from studies in which human subjects, animals, plants, and materials were exposed to smoggy atmospheres or to synthetic mixtures containing oxidant or ozone under controlled conditions. To present convincingly the case that photochemical oxidant air pollution is a problem, it would be necessary and sufficient to present (1) data on the concentration and frequency of occurrence of photochemical oxidants, and (2) evidence regarding the adverse effects of oxidants. Data on the occurrence of oxidants are given in this chapter, but only in a brief summary form, since the occurrence of oxidant at problem levels is well established. Evidence on the effects of oxidant/ozone is presented and discussed in detail in subsequent chapters. For a more detailed discussion of ambient levels (and variations in those levels) of ozone and other oxidants and of some nonoxidant photochemical pollutants, the reader is referred to the literature.[26,27]

Oxidant Concentrations in Urban Atmospheres

Concentrations of oxidant in ambient air were measured in 1974 at some 340 monitoring stations operated by state and local control agencies. Such data are gathered by EPA, stored in EPA's National Air Data Bank, analyzed for trends, and reported in summary form as EPA reports.[28,29,30,31,32]

Table 3-1 shows the maximum hourly average as well as the number of days when the maximum hourly average oxidant concentration was equal to or exceeded 294, 196, and 98 µg/m³ (0.15, 0.10, and 0.05 ppm) for 12 monitoring sites in urban areas. Higher readings of 0.52 ppm in Philadelphia and 0.35 ppm in St. Louis were judged to be spurious and were discarded. More recent data for 16 urban areas are given in Table 3-2.

In general, locations within the Los Angeles Basin urban area have the highest peak oxidant concentrations (represented by the 99th percentile values) as well as the most frequent violations of the oxidant standard. High levels are also observed in the northeast corridor between Washington, D.C., and Boston, Mass. In general, most cities for which extensive ozone data are available have been shown to exceed the national air quality standard for ozone of 160 µg/m³ (0.08 ppm).

A number of these urban areas have experienced very high levels of oxidant, exceeding 600 µg/m³ (0.30 ppm). Los Angeles, for example, recorded maximum 1-hr values in excess of 1200 µg/m³ (0.60 ppm). Denver, Philadelphia, Houston, and the area just east of New York - northeastern

TABLE 3-1. SUMMARY OF MAXIMUM OXIDANT CONCENTRATIONS IN SELECTED URBAN AREAS

Urban area	Year	Total days of valid data	Number of days with at least 1 hourly average equal to or exceeding			Maximum hourly average,	
			98 µg/m³ (0.05 ppm)	196 µg/m³ (0.10 ppm)	294 µg/m³ (0.15 ppm)	ppm	µg/m³
Los Angeles, Calif.	1964-1967	730	540	354	220	0.58	1137
Pasadena, Calif.	1964-1967	728	546	401	299	0.46	902
San Diego, Calif.	1964-1967	623	440	130	35	0.38	745
Sacramento, Calif.	1964-1967	711	443	104	16	0.26	510
Santa Barbara, Calif.	1964-1967	723	510	76	11	0.25	490
San Francisco, Calif.	1964-1967	647	185	29	6	0.22	431
Philadelphia, Pa.	1964-1972	1783	723	186	39	0.33	647
St. Louis, Mo.	1964-1972	2014	1042	152	27	0.22	431
Cincinnati, Ohio	1964-1972	1668	749	105	14	0.26	510
Denver, Colo.	1964-1972	1542	934	179	35	0.36	510
Washington, D.C.	1964-1972	2157	1032	193	25	0.25	490
Chicago, Ill.	1964-1972	2042	828	85	14	0.20	392

TABLE 3-2. OXIDANT CONCENTRATIONS OBSERVED IN SELECTED URBAN AREAS OF THE UNITED STATES, 1974-75

Urban area	Total no. of valid sites[a]	No. of sites exceeding oxidant standard[b]	Range of 2nd max. 1-hr values, $\mu g/m^3$ (ppm)	% of days oxidant standard violated at the worst site
New York, N.Y. - Northeastern N. J.	8	5	259-510 (0.13-0.26)	19.7
Los Angeles - Long Beach, Calif.	3	33	255-784 (0.13-0.40)	38.6
Chicago, Ill. - Northwestern Ind.	6	6	163-427 (0.08-0.22)	8.0
Philadelphia, Pa.	10	10	216-625 (0.11-0.32)	38.2
Detroit, Mich.	2	2	455-514 (0.23-0.26)	3.1
Boston, Mass.	7	7	186-376 (0.09-0.19)	8.2
Washington, D.C.	8	8	363-451 (0.18-0.23)	27.4
Cleveland, Ohio	5	5	245-411 (0.12-0.21)	3.6
Minneapolis - St. Paul, Minn.	2	1	141-206 (0.07-0.10)	4.2
Houston - Galveston, Tex.	4	4	304-588 (0.16-0.30)	23.5
Baltimore, Md.	2	2	314-372 (0.16-0.19)	15.4
Dallas - Fort Worth, Tex.	2	2	274-323 (0.14-0.16)	14.8
Milwaukee - Racine, Wis.	7	7	332-425 (0.17-0.22)	14.5
Seattle - Tacoma, Wash.	4	3	118-235 (0.06-0.12)	7.8
Cincinnati, Ohio - Northern Ky.	6	6	284-412 (0.14-0.21)	23.1
Denver, Colo.	6	6	212-349 (0.11-0.18)	27.5

[a] Only sites having a minimum of 4000 observations were included in this summary.

[b] The oxidant standard is a 1-hr average of 160 $\mu g/m^3$, not to be exceeded more than once per year.

New Jersey have experienced levels in excess of 600 $\mu g/m^3$ (0.30 ppm). Hourly values exceeding 400 $\mu g/m^3$ (0.20 ppm) have occurred in most of the major urban areas.

The data in Table 3-1 and some of those in Table 3-2 were obtained by the potassium iodide method,[26] and therefore they include both ozone and nonozone oxidant species. Data on ozone alone have been obtained only for the more recent years, after the chemiluminescence method for measurement of ozone was introduced. National summaries of such data are not available in a reported form, but detailed tabulations are available.[28,29,30,31,32]

Nonozone oxidants are certain to exist in urban atmospheres, but in concentrations considerably lower than those of ozone.[7,26,27] Measurements of such oxidants in ambient air are scant and consist mostly of PAN data.[21,22] Some recent data on absolute concentrations of PAN and on ozone-to-PAN or oxidant-to-PAN ratios both in urban and rural atmospheres are shown in Tables 3-3 through 3-5.[21] The data currently available indicate, in general, that in urban atmospheres, PAN concentrations are considerably lower than those of oxidant or ozone, but nevertheless, they are not negligible. Hydrogen peroxide (H_2O_2) also has been reported to occur in smog chamber and ambient atmospheres at concentrations as high as 0.18 ppm.[2] Peroxybenzoylnitrate ($C_6H_5COO_2NO_2$) has been reported to occur in urban ambient air in the Netherlands,[23] but no evidence exists for its occurrence in U.S. urban ambient air. The significance of such PAN and other oxidant concentrations can only be assessed on the basis of their adverse effects on humans, vegetation, and materials.

Oxidant Concentrations in Rural Atmospheres

Ozone concentrations in rural atmospheres have been of interest because they were thought to be a measure of the composite contributions of

TABLE 3-3. PAN AND OXIDANT (O_x) MEASUREMENTS (10 a.m. to 4 p.m.), LOS ANGELES, CALIF., 1968[21,a]

No. of samples	PAN conc., ppb	Avg PAN conc., ppb	Range of oxidant conc., ppb	Avg oxidant conc., ppb	Ratio avg O_x/avg PAN	Range of observed O_x/PAN
19	0-10	8.65	30-190	79.0	9.13	5.1-24.4
59	10-20	13.0	44-220	97.0	7.58	3.3-18.3
27	20-30	24.0	70-290	144.0	6.04	2.8-13.0
6	30-40	32.6	100-400	168.0	5.23	3.0-9.7
5	40-50	47.1	160-285	209.0	4.49	3.2-6.1
2	> 50	65.5	243-410	327.0	4.94	3.9-6.0

[a] 1.96 $\mu g/m^3$ = 1 ppb as ozone; 5 $\mu g/m^3$ = 1 ppb as PAN

TABLE 3-4. PAN AND OZONE MEASUREMENTS (10 a.m. to 4 p.m.), HOBOKEN, N.J., 1970[21,a]

No. of samples	PAN conc., ppb	Avg PAN conc., ppb	Range of ozone conc., ppb	Avg ozone conc., ppb	Ratio avg O_3/avg PAN	Range of observed O_3/PAN
14	0-2	1.5	15.0-112.0	42.0	28.0	5.6-77.5
15	2-4	2.8	22.0-150.0	71.0	25.3	10.1-62.5
4	4-6	4.7	100.0-135.0	113.0	24.0	22.9-25.0
8	6-8	7.1	97.0-219.0	162.0	22.8	12.6-34.1
2	8-10	9.9	214.0-278.0	246.0	25.1	20.7-29.6

[a] 1.96 $\mu g/m^3$ = 1 ppb as ozone; 5 $\mu g/m^3$ = 1 ppb as PAN.

TABLE 3-5. PAN, OXIDANT, AND OZONE MEASUREMENTS (10 a.m. to 4 p.m.), ST. LOUIS, MO., 1973[21,a]

No. of samples	PAN conc., ppb	Avg PAN conc., ppb	Range of oxidant conc., ppb	Avg oxidant conc., ppb	Ratio avg O_x/avg PAN	Range of ozone conc., ppb	Avg ozone conc., ppb	Ratio avg O_3/avg PAN
3	0-2	1.7	46-100	80.6	47.5	10-40	28.6	16.3
31	2-4	3.0	46-216	84.6	28.3	24-116	48.1	16.2
60	4-6	5.0	30-250	67.3	13.4	10-140	50.2	10.0
31	6-8	6.8	39-224	68.7	10.1	20-120	50.3	7.4
14	8-10	9.3	40-180	84.6	8.1	10-96	50.2	5.4
5	10-12	10.8	30-90	73.4	6.8	30-85	54.0	5.0
10	> 12	18.6	52-100	85.6	4.6	32-80	59.5	3.2

[a] 1.96 $\mu g/m^3$ = 1 ppb as ozone; 5 $\mu g/m^3$ = 1 ppb as PAN.

natural sources to the ambient ozone problem. Recent studies, however, have established the occurrence of pollutant transport, thus refuting this early notion; and it is now widely accepted that rural areas are not necessarily unaffected by anthropogenic pollution. This conclusion has led air pollution specialists to use the term "rural" to signify areas or atmospheres that are nonurban but are occasionally susceptible to anthropogenic pollutants, and the term "remote" to signify areas or atmospheres so far removed from anthropogenic pollutant sources that their contamination by such pollutants is highly unlikely.

Before 1962, ozone concentrations both in rural and in remote areas appeared to be considerably below the NAAQS level of 160 $\mu g/m^3$ or 0.08 ppm (Table 3-6).[17] Comparable levels were observed also in rural North Carolina during 1964-67.[34] Measurements in the last 5 to 6 years, however, showed that the ozone concentrations in several rural areas exceeded 160 $\mu g/m^3$ (0.08 ppm) with a frequency comparable to or even surpassing that observed in many urban areas.

This fact is illustrated by the data in Table 3-7, which were obtained by recent multiyear field studies sponsored by EPA.[35] Similar data were also reported by New York State investigators.[6] Such increased ozone levels (i.e., >160 $\mu g/m^3$, or >0.08 ppm) in rural areas are almost certain to be caused largely by anthropogenic ozone and/or precursors generated locally or transported into such areas from urban centers.[20,33,35,36,40,43] There is evidence

TABLE 3-6. CONCENTRATIONS OF TROPOSPHERIC OZONE BEFORE 1962[17]

Observer	Location, time, and remarks	Altitude	O_3, $\mu g/m^3$ Range[a]	O_3, $\mu g/m^3$ Average[a]
Cötz and Volz (1951)	Arosa, Switzerland, 1950-51, high valley, daily maximum values	1860 m	19-90	50
Regener (1957)	Mt. Capillo and Albuquerque,	3100 m	18-85	45
	New Mexico, 1951-52	1600 m	3-120	36
Regener (1957)	O'Neil, Nebraska, 1953	12.5 m above ground	30-100	60
Ehmert (1952)	Weissenau, Bodensee, Germany, 1952	20 m	0-90	35
		above ground	0-70	30
Teichert (1955)	Lindenberg Obs., Germany, 1953-54	80 m	0-50	30
		above ground	0-50	27
Kay (1953)	Farnborough, England, 1952-53	0-12,000 m	26-50	38
Brewer (1955)	Tromsö, Norway, 1954	0-10,000 m	60-70	65
Rice and Pales (1959)	Mauna Loa Observatory, Hawaii	3000 m	30-62	45
Wexler et al. (1960)	Little America Station, Antarctica	100 m	20-60	45

[a] As interpreted from the published data. The values sometimes represent absolute maxima, sometimes mean maxima.

TABLE 3-7. SUMMARY OF OZONE DATA FROM 1973-75 OXIDANT STUDIES, RESEARCH TRIANGLE INSTITUTE[35]

Station	Year	Type of station	Average O_3		No. hours ≥0.08 ppm	No. hours	% Hours ≥0.08 ppm
			ppm	$\mu g/m^3$			
McHenry, Md.	1973	Rural	0.074	145	600	1662	37.0
Kane, Pa.	1973	Rural	0.065	127	639	2131	30.0
Coshocton, Ohio	1973	Rural	0.056	110	357	1785	20.0
Lewisburg, W. Va.	1973	Rural	0.0952	187	249	1663	15.0
Wilmington, Ohio	1974	Rural	0.052	102	259	1751	14.9
McConnelsville, Ohio	1974	Rural	0.057	112	262	2011	13.0
Wooster, Ohio	1974	Rural	0.047	92	262	1878	14.0
McHenry, Md.	1974	Rural	0.057	112	262	2011	13.0
DuBois, Pa.	1974	Rural	0.056	110	341	1667	20.5
Canton, Ohio	1974	Urban	0.035	69	148	1829	8.0
Cincinnati, Ohio	1974	Urban	0.025	49	54	1548	3.5
Cleveland, Ohio	1974	Urban	0.031	61	51	1652	3.0
Columbus, Ohio	1974	Urban	0.033	65	113	1935	5.8
Dayton, Ohio	1974	Urban	0.035	69	114	1576	7.2
Pittsburgh, Pa.	1974	Urban	0.028	55	106	1622	6.5
Bradford, Pa.	1975	Rural	0.040	78	100	2332	4.3
Lewisburg, W. Va.	1975	Rural	0.038	74	59	2386	2.5
Creston, Iowa	1975	Rural	0.035	69	17	2117	0.8
Wolf Point, Mont.	1975	Rural	0.028	55	0	2160	0.0
DeRidder, La.	1975	Rural	0.030	59	38	2994	1.3
Pittsburgh, Pa.	1975	Urban	0.030	59	227	2841	8.0
Columbus, Ohio	1975	Urban	0.022	43	43	2885	1.5
Poynette, Wis.	1975	Rural	0.038	74	126	2663	4.7
Cedar Rapids, Iowa	1975	Urban	0.025	49	6	2781	0.2
Des Moines, Iowa	1975	Urban	0.036	70	124	2528	4.9
Omaha, Neb.	1975	Urban	0.035	69	64	1787	3.6
Nederland, Tex.	1975	Urban	0.027	53	138	2714	5.1
Port O'Conner, Tex.	1975	Rural	0.027	53	99	2912	3.4
Austin, Tex.	1975	Urban	0.025	49	19	2504	0.8
Houston, Tex.	1975	Urban	0.026	51	141	2104	6.7

that some rural oxidant is anthropogenic in nature. This evidence is based on measurement of Freons or of acetylene, or on results of wind back-trajectory analysis, both of which are indications of the passage of the rural air over urban sources.[20,33,36,40,43] Additional data on ozone concentrations in remote areas are presented in Chapter 4.

Data on nonozone oxidants in rural or remote atmospheres are scarce. Recent data on PAN in the rural atmosphere at Wilmington, Ohio, showed levels considerably lower than those found in urban atmospheres, both absolutely and relative to ozone concentrations. Earlier as well as recent[2] measurements of PAN and oxidant/ozone showed that (1) the PAN concentrations are much lower than those of oxidant/ozone and (2) the PAN-to-oxidant/ozone ratio varies with location, the rural areas showing lower values than the urban centers.[21] The maximum PAN concentration from 1500 samples taken in August 1974 was 4.1 ppb, with the daily maximum rarely exceeding 3.0 ppb,[21] even though the ozone concentration frequently exceeded 160 $\mu g/m^3$ (0.08 ppm). Such low rural PAN concentrations, expressed in absolute terms as well as in terms relative to ozone, can be explained in different ways. Accordingly, one explanation is that the chemical mechanism of oxidant formation is such that the lower reactant concentrations (of HC and NO_x) and the prolonged irradiation conditions that characterize rural pollutant mixtures result in lower PAN yields and lower PAN-to-ozone product ratios.[21] Another explanation lies in the reversible decomposition of PAN into NO_2 and peroxyacyl radical; in rural areas where NO_2 is nearly absent, PAN decomposition is enhanced.[13] A third conceivable explanation is that the lower PAN-to-ozone ratios in rural atmospheres may reflect a greater proportion of stratospheric ozone relative to the photochemically produced ozone. The explanations based on the chemical mechanism are believed to be the more plausible ones, at least for the Wilmington case.[21] The stratospheric ozone explanation may be a valid one in the cases in which PAN is undetectable.

Patterns of Variation in Oxidant Concentrations

Patterns of variation in oxidant or ozone concentrations are of interest for at least two reasons. They provide a more detailed description of the oxidant problem, which in turn serves to guide the control effort better. Such patterns also help to explain the chemical and physical mechanisms by which emissions of precursors (or of ozone) disperse, react, and ultimately cause the observed oxidant problems.

The most conspicuous variation patterns are the seasonal and diurnal ones. Such patterns result largely from (1) variations in emissions of oxidant-forming pollutants, (2) variations in atmospheric transport and the dilution processes, and (3) variations in other atmospheric variables involved in the photochemical formation of oxidants. Detailed descriptions of the seasonal and diurnal patterns for oxidant/ozone concentrations and some explanatory discussions can be found in the literature.[7,9,26]

Briefly, the striking characteristics of the seasonal and diurnal patterns are (1) the occurrence of higher oxidant concentrations in the summer months, and (2) the occurrence of a daily oxidant peak in the early afternoon hours. Of these, the seasonal pattern is consistent with and, in fact, supports the theory of photochemical formation of atmospheric oxidant in that the higher temperatures and sunlight intensities in the summer enhance the photochemical process.[19]

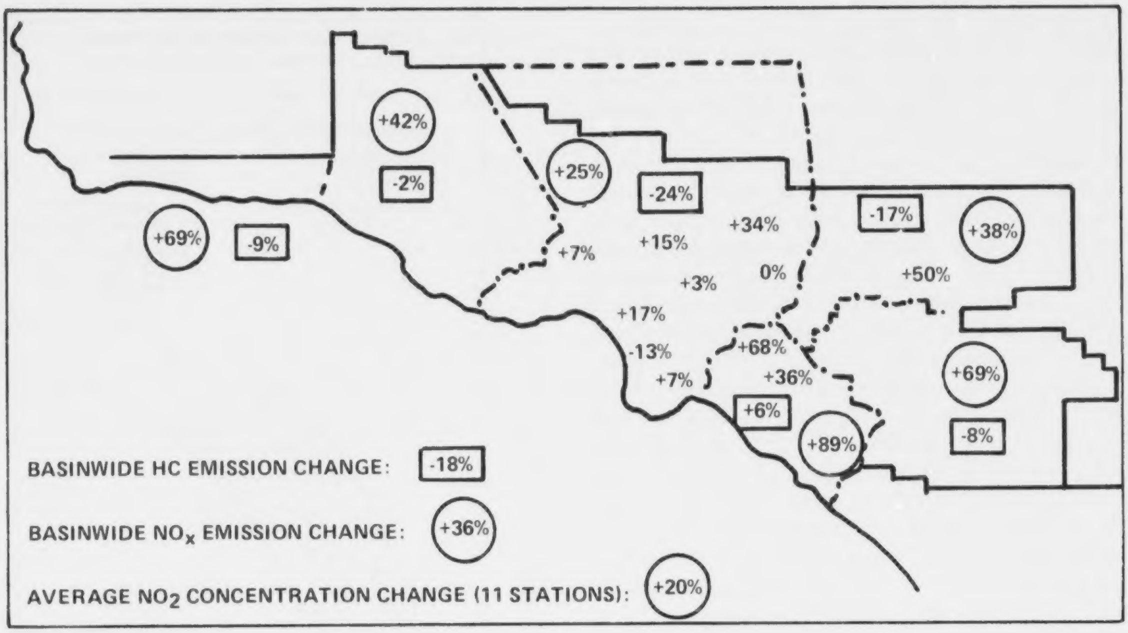
Unlike the seasonal patterns, the diurnal pattern can be explained in more than one way. Until recently, the only explanation accepted was based on the assumption that the daily oxidant peak at a particular location resulted from local emission sources or from those that had been transported to the site. The reactions normally occurred several hours before the oxidant peak.[19]

A more recent theory explains the oxidant peak as the net result of three simultaneously occurring processes:[6,25] (1) downward transport of oxidant/ozone from layers aloft; (2) destruction of oxidant/ozone on surfaces and in reaction with NO at ground level; and (3) photochemical in situ production of oxidant/ozone. Also, interpretation of reported evidence suggests that downward transport and destruction by reaction with NO, rather than photochemical formation,[6,25] determine the concentration of oxidant/ozone observed at ground level for the cores of some cities as well as remote areas. Whether the oxidant/ozone in the layers aloft originates from natural or anthropogenic sources is a question that some investigators consider unresolved;[5,6] the bulk of the recent evidence on long-range pollutant transport, however, points to the anthropogenic origin.[25,31]

ASSOCIATION OF OZONE WITH OTHER CONSTITUENTS OR MANIFESTATIONS OF SMOG

The association of ambient levels of ozone and NO_2 does not show well-defined patterns that would suggest a certain impact of ozone-related control on NO_2. Trend analysis of Los Angeles data shows the ambient NO_2 concentration to increase and ozone to decrease during 1965-74 (see Figures 3-1 and 3-2).[42] These trends have been explained as reflecting the effects of the HC and NO_x emission rate changes during that period. The effect of the ozone-related HC control alone on NO_2 cannot be delineated from the apparently overwhelming effect of the NO_x emission change. A recent study by Trijonis of the ambient NO_2-precursor relationships provided evidence that the hydrocarbon emissions factor had only a small codirectional effect on ambient NO_2, whereas the NO_x factor had a much stronger effect, equivalent to a 1:1 correspondence between NO_x emission change and ambient NO_2 concentration change.[41] Laboratory (smog chamber) data also show (1) a nearly 1:1 correspondence between fractional changes in the NO_x precursor factor and fractional changes in "daily" average or maximum 1-hr NO_2, and (2) a small effect upon NO_2 from change in HC.[8,16]

The association of ozone and PAN has been explored using the limited amount of ambient data available, and some explanations and interpretations have been given of the results. Earlier and recent measurements of PAN and oxidant/ozone showed (1) the PAN concentrations to be much smaller than those of oxidant/ozone, and (2) the PAN-to-oxidant/ozone ratio to vary with location, the rural areas showing lower values than the urban centers.[21] The lower PAN-to-oxidant/ozone ratios in rural areas can be explained mechanistically either as a result of enhanced PAN decomposition in NO_2-free atmospheres or as a result of the relatively lower concentrations and prolonged irradiation conditions, as a result of pollutant transport, that characterize the reaction systems in rural atmospheres. Overall, the ambient data associations do not provide conclusive evidence on the impact of ozone-related control on PAN. The evidence, however, from laboratory and theoretical studies is definitive and shows that for urban atmospheres, changes in HC and NO_x


+42%
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+50%
+17%
-13%
+68%
+7%
+36%
+69%
+6%
+89%
-8%
BASINWIDE HC EMISSION CHANGE: -18%
BASINWIDE NO$_X$ EMISSION CHANGE: +36%
AVERAGE NO$_2$ CONCENTRATION CHANGE (11 STATIONS): +20%

Figure 3-1. Trends in NO_2 air quality and in HC and NO_x emission. Los Angeles Basin, 1965-74.

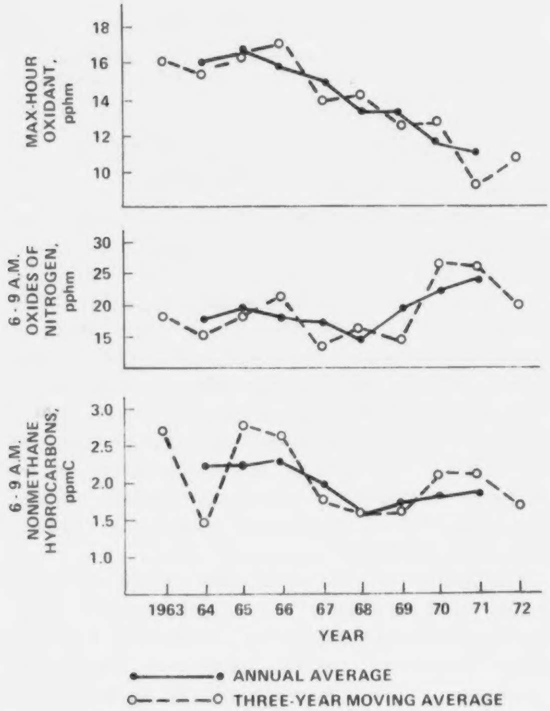

Figure 3-2. Pollutant trends in Los Angeles, annual and 3-year moving averages.[42]

emissions should have greater impacts on ambient PAN than on ambient ozone.[8]

Ambient aldehydes, although nonoxidants, are also of interest here because they are constituents of the photochemical pollution complex and are known to have some physiological effects (Chapters 8 and 9). However, aldehydes are also directly discharged into the atmosphere as automotive emissions.[10,39] Concentrations in urban atmospheres seem to rise quickly in the early morning hours, reach and maintain a broad plateau through most of the day, and then decrease in the afternoon.[7] This pattern probably reflects the dual origin (primary and secondary) of the ambient aldehydes as well as the fact that rapid photochemical formation of aldehydes, unlike oxidant/ozone, begins with the onset of irradiation.[19] Ambient data associations do not provide evidence on the impact of ozone-related control on aldehydes; laboratory data, however, do suggest a nearly 1:1 correspondence between fractional changes in HC and fractional changes in ambient (photochemical) aldehydes.[8]

The association of ambient oxidant/ozone with eye irritation has been explored extensively, but only in one location, the Los Angeles Basin. This association has been described in detail in the predecessor of this criteria document and need not be elaborated here. Briefly, ambient oxidant/ozone appears to correlate well with eye irritation, a relationship which, however, is clearly not of cause-effect nature. Lack of causative relationship means that control of ozone is not expected to have a direct effect on eye irritation. However, the high degree of correlation between the ambient data, the laboratory evidence of good correlation between the photochemical process and eye irritation,[45] and, finally, the fact that some eye irritants, (namely formaldehyde, acrolein, and PAN) are known to be products of atmospheric photo-oxidation of HC, when considered together all suggest that oxidant-related emission control will probably have a reducing effect on eye irritation for Los Angeles. Eye irritation has been observed also in other areas. A pollution episode, for example, in New York City during November 23-25, 1966, caused numerous eye irritation complaints from residents.[1] Oxidant/ozone measurements were not made during the episode, and therefore an association could not be established. The problem could have been caused either by primary pollutants (aldehydes) mainly, or by both primary and secondary pollutants. In conclusion, based on present-day knowledge and understanding, the evidence and conclusions obtained for the Los Angeles area cannot be assumed to have universal validity. For such assumption to be supported (or refuted), additional evidence must be obtained.

The association of ambient ozone with visibility or visibility-reducing aerosol is perhaps the most interesting of the associations discussed here. Public reaction to the visibility problem has been stronger than to the ozone problem, and there is a feeling among local air pollution control officials that the often upsetting ozone control programs will be more acceptable if they are shown to have a beneficial impact on the haze problem also. In spite of this strong interest in the ozone-haze association, relatively little evidence is currently available on this subject. The evidence available is mostly on the Los Angeles atmosphere. A considerable part was obtained in the course of conducting the California Air Characterization Study (ACHEX)[14] and has been summarized informatively in a recent National Academy of Sciences report.[42] The part of the evidence of most interest here is shown in Figure 3-3 in terms of diagrams indicating a good correlation for the Los Angeles Basin between visibility reduction and ambient ozone concentration. Somewhat similar, but weaker, evidence was reported by Husar et al.[15] and by Wolff et al.[46] who showed that synoptic-scale areas with elevated ozone concentrations roughly corresponded in location and magnitude with areas of reduced visibility ("haze blobs"). Such good correlations between ozone and haze are self-explanatory in the cases in which the haze-causing aerosols consist largely of photochemical reaction products. This clearly is the case with Los Angeles, as deduced from the findings that the optically active aerosol in the Los Angeles air consists primarily of photochemically generated sulfate with lesser amounts of nitrates and organics.[3,11,12,44]

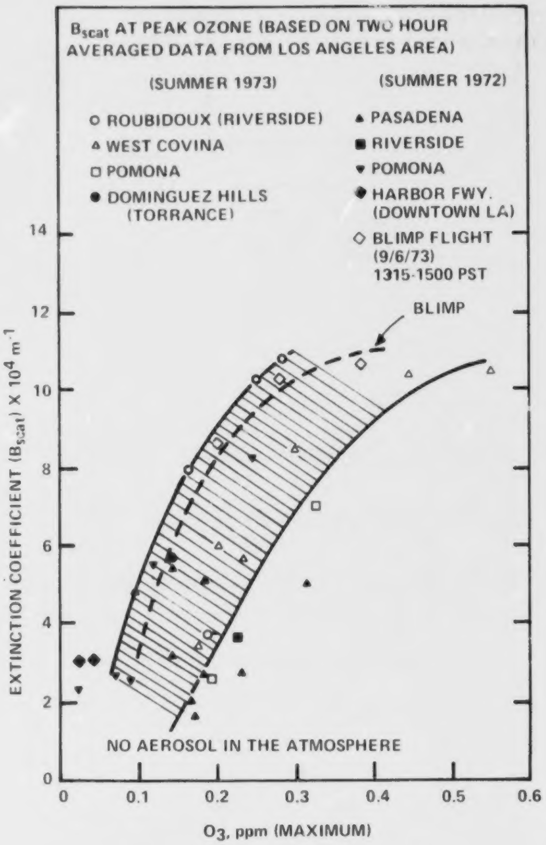

Figure 3-3. Correlation between B_{scat} and maximal ozone concentration.[15]

The good ozone-haze correlation observed in Los Angeles, the aerosol composition data, the good correlation between haze and ambient sulfates,[24] and, finally, the experimental and theoretical evidence on the atmospheric photo-oxidation process[18,3] all point to the conclusion that oxidant-related control should also cause some reduction in haze in Los Angeles. This conclusion, with two important qualifications, is probably correct. The qualifications are that (1) ozone-related control, although beneficial, is not necessarily the most effective means for controlling haze, and (2) the Los Angeles evidence on the impact of ozone-related control on haze does not have universal validity. The second qualification is supported by the case of Denver, for example, where the haze problem appears to be caused by primary aerosols.[38,4] Such aerosols cannot possibly be impacted by the ozone-related control except, of course, when HC emission control measures happen to reduce vehicular aerosol emissions (e.g., measures reducing automobile traffic). Houston also has been reported to show interpollutant relationships different from those occurring in Los Angeles.[4]

SUMMARY

Photochemical oxidants are products of atmospheric reactions involving organic pollutants, NO_x, oxygen, and sunlight. They consist mostly of ozone, NO_2, and PAN, with smaller amounts of other peroxyacylnitrates and other peroxy-compounds, and are formed along with other photochemical products such as aldehydes, gaseous and particulate nitrates, and sulfates. They originate mainly from volatile organic and NO_x emissions associated with human activities. Photochemical oxidant formation is a complex function of emissions and meteorological patterns.

Peak concentrations of oxidant expressed as ozone are generally higher within urban and suburban areas, reaching levels in excess of 590 $\mu g/m^3$ (0.3 ppm). In rural areas, peak concentrations are lower but often exceed the 1-hr National Ambient Air Quality Standard of 160 $\mu g/m^3$ (0.08 ppm). Dosages or average concentrations, however, are comparable to or even higher than those in urban areas. Because of pollutant transport, the oxidant problem is viewed as having regional dimensions in the hundreds-of-miles range.

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4. SOURCES AND SINKS OF OXIDANTS

PHOTOCHEMICAL FORMATION OF OXIDANTS

Introduction

All the evidence presently available indicates that in the urban centers and adjacent downwind areas where severe oxidant problems occur, the major cause by far is photochemical oxidant formation. In areas with less severe problems, and especially in remote areas, nonphotochemical sources (e.g., stratospheric ozone intrusion) may be significant and on occasions may be the dominant sources.

When photochemical oxidants in air were identified as products of a photochemical process involving primary air pollutants, it became immediately apparent that the problem could not be abated by traditional methods of direct control. The chemistry of this process had to be clearly understood before rational abatement measures could be devised. Accordingly, in the ensuing years, numerous studies of the oxidant chemistry were conducted with the following specific objectives:

1. To identify the precursors of photochemical oxidants (that is, those primary pollutants that participate as reactants in the oxidant-forming process) for the purpose of directing the control effort to the right targets.
2. To determine the kinetics of the precursor reactions mainly for the purpose of deducing the impact of precursor control on oxidant formation.
3. To determine the stoichiometry of the precursor reactions for the purpose of more fully assessing the pollution problems caused by such precursors.
4. To elucidate in detail the oxidant-forming mechanism for the purpose of providing deterministic, as distinct from empirical, input to the oxidant abatement effort, and also for the purpose of relating oxidant formation to other manifestations of photochemical pollution.

Several of these objectives have been achieved, at least to the degree of completion such that the information generated was sufficiently comprehensive and reliable to permit development of a crude but promising oxidant abatement strategy. These chemical studies clearly are relevant to the purposes of this document; however, they need not all be included in this review or expanded on in detail except where they pertain to applications directly related to the questions of oxidant control.

To serve the purposes of this document better, the discussion of the chemistry pertaining to photochemical oxidants will be divided and presented in several sections. The following section on atmospheric reaction mechanisms includes a general discussion of the chemical processes and a review of recent atmospheric chemistry studies, focusing only on those findings that are judged to have had the strongest impact on the understanding of the oxidant formation mechanism. It is intended and hoped that this introductory discussion and review will prepare the reader to understand better the subsequent sections dealing with the specific applications of the information used in formulating oxidant control strategies. In those sections, the reaction mechanisms and other pertinent aspects of the oxidant chemistry will be discussed critically and more comprehensively. For additional detailed discussions of the earlier studies of atmospheric chemistry, the reader is referred elsewhere.[54,78,92,93]

Finally, there is one important difference in emphasis between the following reaction mechanism section and the preceding chapters. In the preceding chapters, ozone and other oxidants were dealt with nearly always collectively, using the term "oxidant." In the discussions of reaction mechanisms, however, all mechanistic interpretations regarding oxidant, in actuality, pertain to ozone alone. Analogous mechanistic discussions addressed to other oxidants will not be included in this document mainly because the emphasis is on ozone and because the mechanistic information available for other oxidants is considerably deficient.

Atmospheric Reaction Mechanisms

The pioneering work of Haagen-Smit in 1952[54] first demonstrated through laboratory experimentation that the photochemical oxidants present in an urban atmosphere may indeed be products of atmospheric photochemical reactions involving organic (e.g., hydrocarbons) and inorganic (e.g., nitrogen oxides) pollutants. Since then, numerous studies of oxidant formation have been conducted,[4,5,78,93] and a wealth of information is now available regarding the stoichiometry, kinetics, and mechanisms of these HC-NO_x-air-sunlight reactions. The following discussion summarizes the results of these studies and presents highlights of the chemical mechanism that is presently thought to explain best the phenomenon of photochemical oxidant formation.

All experimental reserach concerned with the mechanism of atmospheric oxidant formation was conducted in the laboratory using experimental conditions that were similar to but not nearly as complex as those prevailing in the ambient atmosphere. Such simplifications of the natural system were necessary to facilitate research, but they also limited the validity of extrapolating research findings to ambient conditions. Thus most mechanistic evidence obtained to date pertains solely to the oxidant formation process occurring in laboratory systems. The most that can be said at this time is that all of the reaction steps known to occur in the experimental reaction systems are almost certain to occur in the ambient atmosphere also, although perhaps with varying significance. The converse, however, may not necessarily be true: that is, unidentified reaction steps may occur in the ambient atmosphere but not in laboratory simulations.

The overall chemical changes that are observed to occur when a mixture of HC and NO_x pollutants is exposed to sunlight under conditions similar to those in the atmosphere are illustrated in Figure 4-1. The curves of Figure 4-1 depict the overall photochemical process as consisting of two distinct reaction stages occurring consecutively. During the first stage, nitric oxide (NO) is converted into nitrogen dioxide (NO_2) without any appreciable buildup of ozone (O_3) or other non-NO_2 oxidants. The second stage starts when almost all of the HO has been converted into NO_2 and is characterized by rapid accumulation of ozone and other oxidant and nonoxidant products.

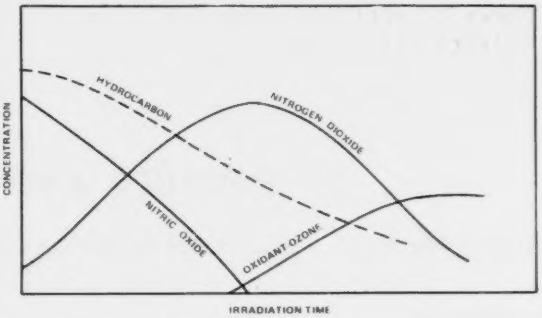


Figure 4-1. Chemical changes occurring during photoirradiationof hydro-carbon-nitrogen oxide-air systems

Several mechanisms explaining the observations depicted in Figure 4-1 have been postulated. Although these mechanisms differ substantially in chemical detail, they all explain ozone formation in the atmosphere to be the net result of the following main reactions:

$$NO_2 \xrightarrow[\text{light}]{UV} NO + O \qquad (4\text{-}1)$$

$$O + O_2 \xrightarrow{M} O_3 + M \qquad (4\text{-}2)$$

$$O_3 + NO \longrightarrow NO_2 + O_2 \qquad (4\text{-}3)$$

$$XO_2 + NO \longrightarrow NO_2 + XO \qquad (4\text{-}4)$$

$$2NO + O_2 \longrightarrow 2NO_2 \qquad (4\text{-}5)$$

where M is the third body in a reaction, and X is hydrogen (H) or organic radical (R or RCO). Through this mechanistic procedure, the NO product from the photolysis of NO_2 reacts rapidly with and consumes ozone to regenerate the photolyzed NO_2. Therefore, unless other processes convert the NO into NO_2, ozone is not allowed to accumulate to significant levels. However, other NO conversion processes do exist—mainly the reactions of NO with XO_2 and, to a much lesser degree, with molecular oxygen (O_2). The reaction with XO_2 occurs only when photochemically reactive organic compounds are present, in which case the reaction can be sufficiently rapid to cause atmospheric accumulation of ozone to significant levels. In the absence of reactive organics, the only NO conversion process parallel to the O_3-NO reaction is the reaction of NO with O_2; this reaction, however, is relatively slow and does not cause significant ozone accumulation.

In mathematical terms, the ozone buildup in the atmosphere obeys the equation[28]

$$[O_3] = \frac{[NO_2]}{[NO]} \qquad (4\text{-}6)$$

where I is light (sunlight) intensity, and k is a constant. This equation is derived by applying the steady state hypothesis to chemical reaction steps 4-1, 4-2, and 4-3. This equation illustrates the effect on ozone buildup of any process that converts NO into NO_2 (e.g., the reaction of NO with XO_2); such effect is toward high $[NO_2] : [NO]$ ratios, and hence high levels of ozone buildup. Ambient measurements in downtown Detroit[132] in the summer of 1973 and from the Los Angeles Reactive Pollutant Project (LARPP)[18] have essentially verified the photostationary state equation in tie atmosphere within the stochastic limits of the turbulent atmosphere.[124]

Though the overall mechanistic scheme depicted by processes 4-1 through 4-5 was established long ago, the detailed reactions explaining the consumption of the organic and inorganic reactants and the formation of the XO_2 radicals (e.g., reaction 4-4) have been relatively obscure. It is in this latter mechanistic area that some important developments have taken place in the past 5 to 6 years. These new findings and their impact on current understanding are summarized here. The significance of these findings in the specific applications of reaction mechanisms in the development of mathematical models will be discussed in a subsequent section of this document.

One important accomplishment in recent years in the area of atmospheric reaction mechanisms is the development of computer techniques for simulating the atmospheric smog-forming process.[19,40] Such techniques have provided a useful tool for selecting those reactions that play a key mechanistic role and for predicting the existence of potentially important but as yet unidentified reaction products in the ambient atmosphere.

The most important mechanistic finding, however, in recent years pertains to the identities, sources, and roles of the radicals responsible for the oxidation of the organic and inorganic reactants. Unlike the early emphasis on atomic oxygen, $O(^3P)$, and ozone roles, the current thinking attributes to the hydroxyl radical (OH) most of the hydrocarbon- and aldehyde-consuming chemical activity.[58,133]

Other radicals also, most notably atoms and RO_2, can attack and consume the organic reactant significantly, as illustrated by the data in Table 4-1. These data, derived from a computer simulation of an irradiated *trans*-2-butene/NO_x reaction system, represent calculated rates of attack of several reactive species on butene, at several irradiation times.

TABLE 4-1. CALCULATED RATES OF ATTACK ON *TRANS*-2-BUTENE BY VARIOUS REACTIVE SPECIES IN SIMULATED SMOG SYSTEM AT SEVERAL IRRADIATION TIMES[a]

	Attack rate, ppb/min		
Species	2 min	30 min	60 min
O	0.013	0.018	0.011
O_3	0.026	0.16	0.16
OH	1.72	0.55	0.27
HO_2	0.16	0.15	0.09
NO_3	0.05×10^{-4}	2.2×10^{-4}	2.6×10^{-4}
$O_2\ (\alpha'\Delta)$	2.9×10^{-6}	2.1×10^{-6}	1.3×10^{-6}

[a] Data from Demerjian et al.[40] Initial conditions: [NO], 0.075 ppm; $[NO_2]$, 0.025 ppm; [*trans*-2-butene], 0.10 ppm; [CO], 10 ppm; $[CH_2O]$, 0.10 ppm; $[CH_3CHO]$, 0.06 ppm; $[CH_4]$, 1.5 ppm; relative humidity, 50%.

The radicals OH, HO_2, and XO_2 have also been identified as having major roles in the oxidation of NO into NO_2 (reaction 4-4).[93] Sources of these important radicals are now believed to be the reactions:

$$\text{(Aldehydes, HONO, } O_3\text{, others)} \xrightarrow{h\nu} \text{OH, } HO_2\text{, } RO_2\text{, others} \qquad (4\text{-}7)$$

Oxidation of NO into NO_2 occurs via a chain mechanism illustrated by the following sequence:

$$\text{OH + (hydrocarbons, aldehydes)} \longrightarrow HO_2\text{, } RO_2\text{, others} \qquad (4\text{-}8)$$

$$HO_2 + NO \rightarrow NO_2 + OH \qquad (4\text{-}9)$$

$$RO_2 + NO \rightarrow RO \qquad (4\text{-}10)$$

$$RO + O_2 \rightarrow HO_2 + \text{aldehyde} \qquad (4\text{-}11)$$

Such chain processes are terminated when radicals react to form more stable products, for example:

$$OH + NO_2 \rightarrow HONO_2 \qquad (4\text{-}12)$$

$$HO_2 + HO_2 \text{ (or } RO_2) \rightarrow H_2O_2 \text{ (or ROOH)} \qquad (4\text{-}13)$$

$$HO_2 + NO_2 \rightarrow \text{HONO (or } HOONO_2?) \qquad (4\text{-}14)$$

$$RC(O)O_2 \rightarrow RC(O)O_2NO_2 \text{ (i.e., PAN)} \qquad (4\text{-}15)$$

$$HO_2\text{, } RO_2 + \text{surface} \rightarrow (HO_2\text{, } RO_2) \text{ surface} \qquad (4\text{-}16)$$

A more complete picture of the reactions accounting for the organic oxidant degradation, oxidation of NO into NO_2, and organic product formation in the ambient atmosphere, is illustrated in Figure 4-2 for the case in which the organic reactant is an olefin.[40] Comparably complete mechanistic information is now available for paraffins also, but not for aromatic hydrocarbons.

In the inorganic part of the atmospheric reaction mechanism, some steps recently have become the subjects of controversy because of their different roles in the ambient atmosphere and in the laboratory systems. Thus, the reactions

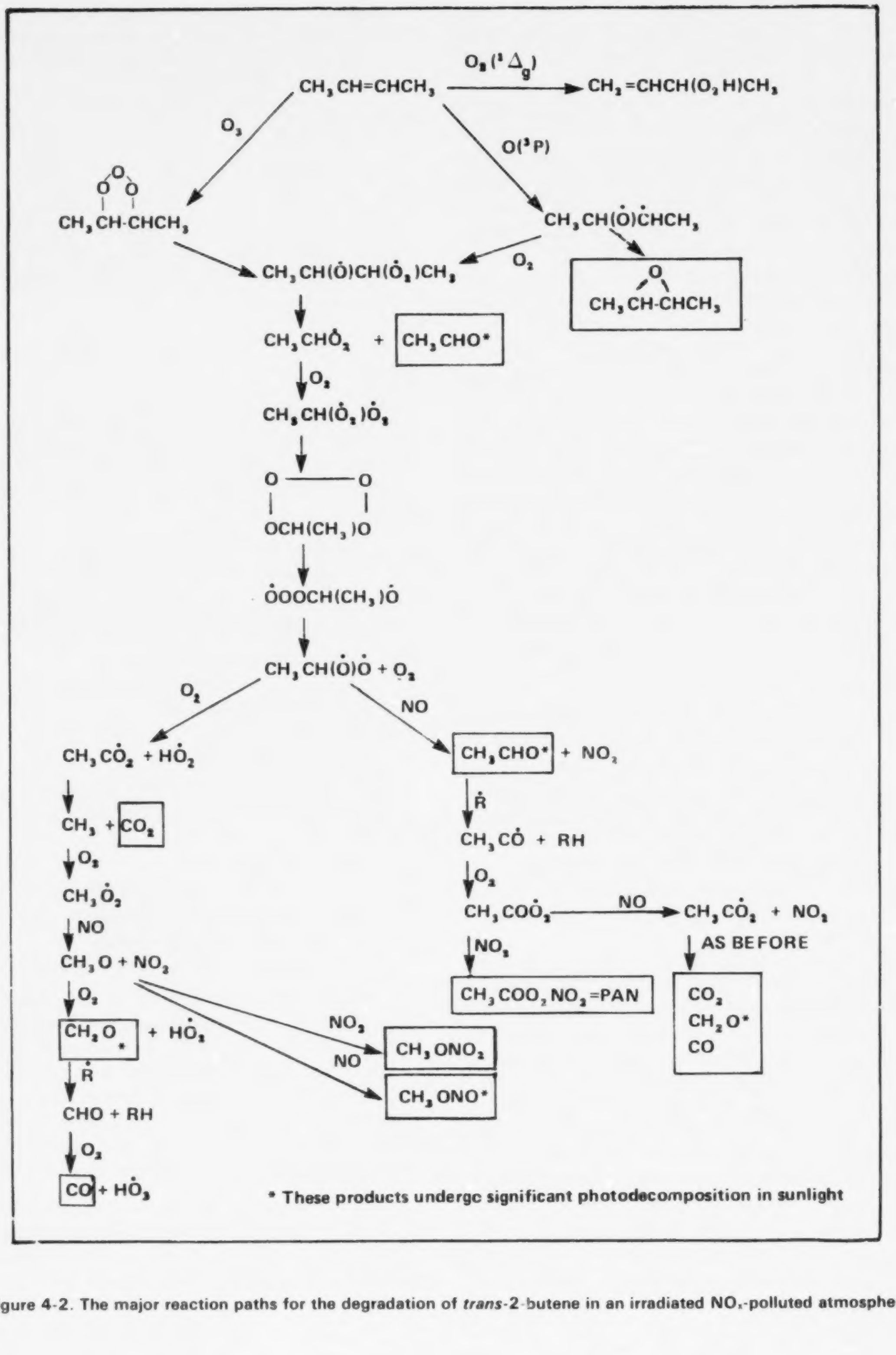
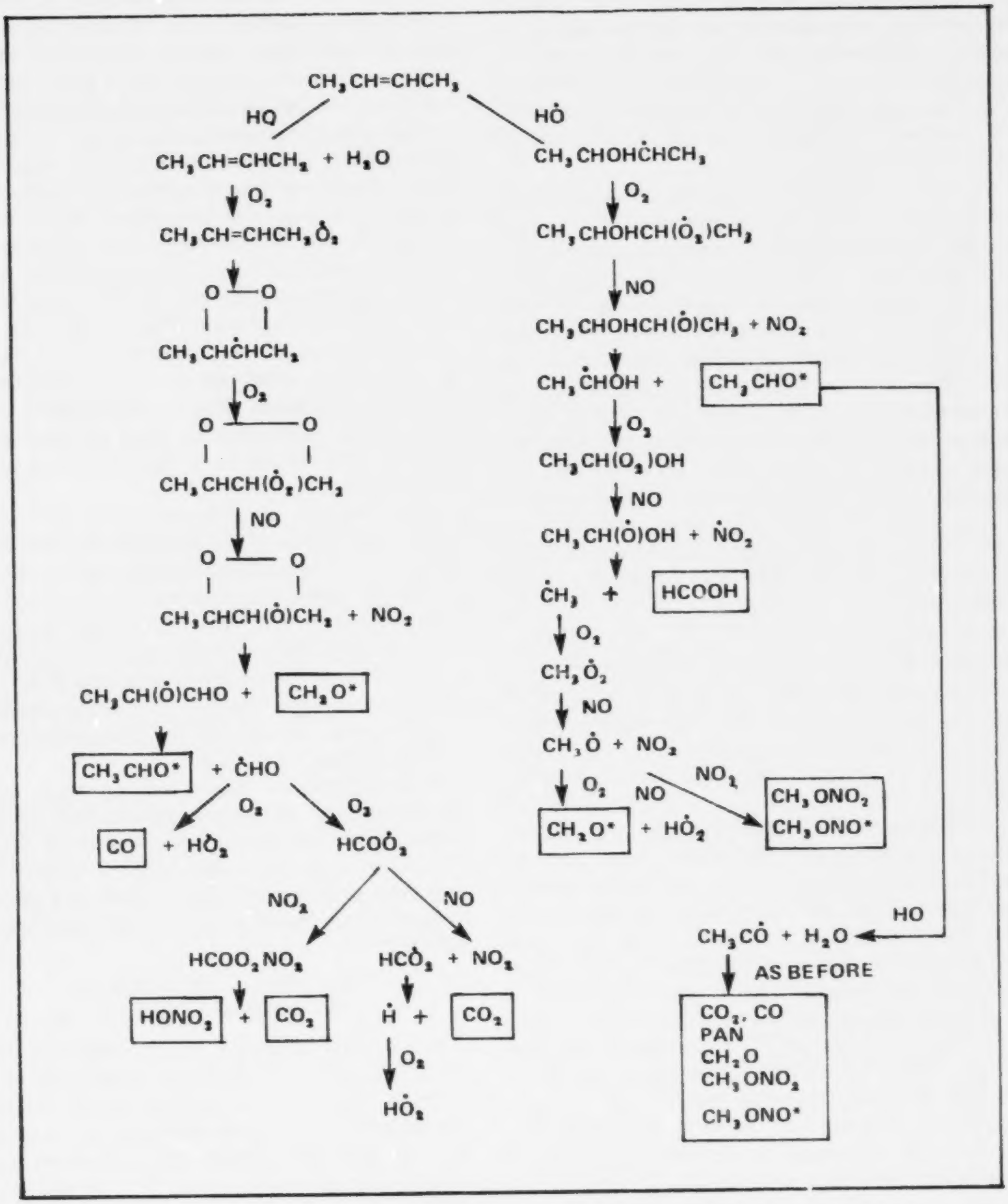

CH$_3$CH=CHCH$_3$
O$_2$($^1\Delta_g$)
CH$_2$=CHCH(O$_2$H)CH$_3$
O$_3$
O(^{3}P)
CH$_3$CH-CHCH$_3$
CH$_3$CH(Ȯ)ĊHCH$_3$
CH$_3$CH(Ȯ)CH(Ȯ$_2$)CH$_3$
O$_2$
CH$_3$CH-CHCH$_3$
CH$_3$CHȮ$_2$ + CH$_3$CHO*
O$_2$
CH$_3$CH(Ȯ$_2$)Ȯ$_2$
O — O
OCH(CH$_3$)O
ȮOOCH(CH$_3$)Ȯ
CH$_3$CH(Ȯ)Ȯ + O$_2$
O$_2$
NO
CH$_3$CȮ$_2$ + HȮ$_2$
CH$_3$CHO* + NO$_2$
CH$_3$ + CO$_2$
Ṙ
CH$_3$CȮ + RH
O$_2$
CH$_3$Ȯ$_2$
CH$_3$COȮ$_2$
NO
CH$_3$CȮ$_2$ + NO$_2$
NO
NO$_2$
AS BEFORE
CH$_3$O + NO$_2$
CH$_3$COO$_2$NO$_2$=PAN
CO$_2$
CH$_2$O*
CO
CH$_2$O* + HȮ$_2$
NO$_2$
CH$_3$ONO$_2$
NO
CH$_3$ONO*
CHO + RH
CO + HȮ$_2$
* These products undergo significant photodecomposition in sunlight

Figure 4-2. The major reaction paths for the degradation of *trans*-2-butene in an irradiated NO$_x$-polluted atmosphere.



CH3CH=CHCH3
HO
HO
CH3CH=CHCH2 + H2O
CH3CHOHCHCH3
O2
O2
CH3CH=CHCH2O2
CH3CHOHCH(O2)CH3
O—O
CH3CHCHCH2
NO
CH3CHOHCH(O)CH3 + NO2
CH3CHOH +
CH3CHO*
O2
O—O
CH3CHCH(O2)CH3
O2
CH3CH(O2)OH
NO
NO
CH3CH(O)OH + NO2
O—O
CH3CHCH(O)CH3 + NO2
CH3 + HCOOH
O2
CH3O2
NO
CH3CH(O)CHO + CH2O*
CH3O + NO2
CH3CHO* + CHO
O2 O2
O2 NO
NO2
CH3ONO2
CH3ONO*
CO + HO2
HCOO2
CH2O* + HO2
NO2 NO
HO
CH3CO + H2O
AS BEFORE
HCOO2NO2
HCO2 + NO2
HONO2 + CO2
H + CO2
O2
HO2
CO2, CO
PAN
CH2O
CH3ONO2
CH3ONO*

Figure 4-2. (Continued). The major reaction paths for the degradation of *trans*-2-butene in an irradiated NO_x-polluted atmosphere.

$$NO + NO_2 + H_2O \rightarrow HONO \tag{4-17}$$
$$N_2O_5 + H_2O \rightarrow 2HNO_3 \tag{4-18}$$

are known to occur both homogeneously (in the gas phase) and heterogeneously. Both reactions are thought to be of relatively little importance in the ambient atmosphere but are suspected to occur significantly on the reactor walls in laboratory systems.[17,71] Questions are thus raised about the applicability of the laboratory findings to the ambient atmosphere, which is an issue of crucial importance.

Another noteworthy finding is that some nitrogenated products of the photochemical smog system, such as PAN and possibly peroxynitric acid ($HOONO_2$) have a greater mechanistic role than thought earlier. Recent investigations of PAN chemistry [34,60,98] have revealed that PAN can thermally decompose to an acylperoxy radical and NO_2. The rate of decomposition is extremely temperature dependent. Because of this temperature dependency, significant levels of PAN can build up early in the day when temperatures are relatively low. In the late afternoon when ambient temperatures are higher, the decomposition of PAN can proceed at a rapid rate, liberating NO_2 molecules that can lead to enhanced ozone production.

The discovery that PAN can thermally decompose and enhance ozone formation suggests that there may be other peroxynitrates that can also affect the rate of smog formation. Recently, peroxynitric acid ($HOONO_2$) was identified by Fourier-transform infrared spectroscopy (FS)[79,95] as an intermediate of photochemical smog systems. This species and the related peroxynitrates ($ROONO_2$) could act as radical sinks and affect the rate of smog formation. The important HO_2 and RO_2 free radicals, as well as NO_2, could be temporarily stored as peroxynitrate and then released (through thermal decomposition of the peroxynitrate) at later stages in the reaction to enhance ozone production. However, recent experimental evidence[33,50,125] indicates that the decomposition of $HOONO_2$ proceeds so rapidly at room temperature that this species will not be a significant sink for HO_2 or NO_2. Consequently, $HOONO_2$ is not likely to be of significance in photochemical smog formation.

This role of the peroxynitrate products raises the more general question regarding the role of atmospheric reaction products in the chemistry of polluted air masses irradiated for several days. Such multiday reaction of pollutant mixtures is now known to occur in the ambient atmosphere and specifically, within urban air masses subjected to long-range transport.[136] While the chemical mechanism of such aged systems cannot be different from that of fresh systems in terms of fundamental reaction steps involved, the relative rates of such steps may be sufficiently different that the relative roles of the organic and NO_x reactants, as well as the relative reactivities of the various organics in producing oxidant/ozone, may be substantially different. Evidence attesting to such differences has recently been reported by researchers who did laboratory and computer simulation studies of fresh and aged HC-NO_x reaction systems. Thus smog chamber studies have suggested that the oxidant-to-precursor dependencies in aged systems may be different in magnitude, and possibly in direction also, from the dependencies observed in fresh systems.[41,71] Current knowledge and understanding of the atmospheric chemistry of aged polluted air are considerably inferior to those pertaining to fresh atmospheres.

The chemical transformation of SO_2 to particulate sulfates is of considerable interest, both in terms of understanding SO_2 removal mechanisms in the atmosphere as well as the potential health and welfare effects[96] associated with particulate sulfates. Though it is beyond the scope of this document to provide a detailed review of the state-of-the-art of SO_x chemistry, a short discussion regarding its interaction in the photochemical process seems warranted.

In recent years, significant progress toward understanding the homogeneous SO_2 oxidation pathways in the atmosphere has been made.[16,119,122] One of the more important contributions has been the experimental gas kinetic rate constant determinations for the HO_2 and HO reactions with SO_2: [20,99]

$$HO_2 + SO_2 \rightarrow HO + SO_3$$
$$HO + SO_2 + M \rightarrow HOSO_2 + M$$

The quantification of these reaction rates in conjunction with theoretical estimates of the reaction rates for the organic radical analogies RO_2 and RO have explained major portions of the SO_2 transformation observed in both experimental smog chamber studies and field investigations.[16,119,142]

In the presence of water vapor, SO_2 reacts rapidly to form H_2SO_4. The details of the reaction pathway for the $HOSO_2$ species is currently unknown, but evidence exists indicating that

H_2SO_4 is the resulting end product. In the atmosphere, H_2SO_4 may undergo complex interactions with aerosol particulate or react with gaseous ammonia to form a host of sulfate salts. Characterization of these condensable species via experimental and ambient observations will be a major area of continued research and will play a significant role in elucidating the mechanistic details of atmospheric SO_x.

Outside the reaction mechanism area, some significant developments have taken place in recent years in the area of reaction product identification. Table 4-2 lists gaseous products and typical concentrations at which such products were found to occur in the ambient atmosphere. Table 4-3 lists gaseous products either observed in laboratory (smog chamber) systems or unreported yet but theoretically expected to exist in the ambient atmosphere. Particulate products also exist but are not easy to identify. Such products are certain to include nitrates and a variety of organic compounds. Tables 4-4 and 4-5 list some reaction products identified in ambient atmosphere aerosols.[93] The significance of these findings is that they provide a more complete description of the photochemical pollution problem and that they verify or further elucidate the atmospheric reaction mechanism.

TABLE 4-2. COMPOUNDS OBSERVED IN PHOTOCHEMICAL SMOG

Compound	Typical (or maximal) concentration reported, ppm	
Ozone, O_3	0.1	(0.6)
PAN, $CH_3COO_2NO_2$	0.005	(0.2)
Hydrogen peroxide, H_2O_2		(0.18)
Formaldehyde, CH_2O	0.04	(0.16)
Higher aldehydes, RCHO		(0.36)
Acrolein, CH_2CHCHO		(0.011)
Formic acid, HCOOH		(0.05)

TABLE 4-3. COMPOUNDS THAT MAY BE FORMED IN PHOTOCHEMICAL SMOG

Compound	Possible synthesis	Reference
Peroxybenzoylnitrate, $C_6H_5COO_2NO_2$	$\phi COO_2 + NO_2$	62
Nitric acid, $HONO_2$	$NO_2 + OH$	55
	$N_2O_5 + H_2O$	91
Organic hydroperoxides, ROOH	$RO_2 + HO_2$	40
Organic peracids, $RCOO_2H$	$RCOO_2 + HO_2$	40
Organic peroxynitrates, RO_2NO_2	$RO_2 + NO_2 + M^a$	40
Ozonides, O_3-olefin	O_3 + olefin + M^a	7
Ketene, CH_2CO	O_3 + olefin	88
Nitrous acid, HONO	OH + NO	8,32
Pernitric acid, HO_2NO2	$NO_2 + HO_2 + M^a$	50,79,95
Pernitrous acid, HO_2NO	$NO + HO_2 + M^a$	31
Organic nitrates, $RONO_2$	$RO + NO_2$	37
	$RO_2 + NO$	37

[a] M represents any molecule that takes part in the three-body process.

In conclusion, the information generated in the years since the issuance of the preceding criteria document provides a much more complete picture of the atmospheric oxidant formation process. Nevertheless, it should be stressed that the present situation is somewhat uncertain because most of the supporting evidence was obtained from laboratory atmospheres and is therefore mainly applicable to such an environment. The mechanism of the oxidant-forming process in the ambient atmosphere, containing a multitude of reacting pollutants, may include reaction steps in addition to those presently recognized. Such additional reactions, for example, known or suspected to occur in the ambient atmosphere, are those causing degradation of aromatic hydrocarbons and of numerous organics into gaseous and particulate products. To date, these degradations are largely unexplored. Important reactions may also be occurring through energy transfer processes promoted by pollutants or other molecules capable of absorbing solar energy and of transferring such energy to nonabsorbing pollutants. Also, the nature and importance of heterogeneous reactions occurring on the surface of ambient aerosol particles[49] and on the surface of laboratory chambers[17] have been explored but are not well understood. Further studies are needed to ascertain the applicability of laboratory data to ambient atmospheres.

Effects of Meteorological Factors

INTRODUCTION

The photochemical oxidant/ozone concentrations observed in the ambient air above urban and nonurban areas are the net result of two broadly defined processes: First, a physical process involving dispersion and transport of the oxidant-precursor emissions; and second, a chemical process involving reaction of the dispersed pollutants under the stimulus of sunlight. The potential effects of meteorological factors on both these processes are obvious. Thus the factors related to atmospheric dilution and transport affect ambient levels of pollutant accumulation and the geographical relationship between source areas and corresponding oxidant problem areas. Solar radiation and ambient temperature are also important by virtue of their effects on the chemical

TABLE 4-4. SECONDARY ORGANIC AEROSOLS[2]

Compounds identified[a]		Possible gas-phase hydrocarbon precursors
Aliphatic multifunctional compounds:		
1. X-$(CH_2)_n$-Y (n=3,4,5):		1. Cyclic olefins
X	Y	
COOH	CH_2OH	$(CH_2)_n$ CH CH
COOH	COH	
COOH	COOH	
$COOH_b$	CH_2ONO	
or COH	CH_2ONO_2	
COH	CH_2OH	
COH	COH	and/or diolefins
$COOH_b$	COONO	
or COH	$COONO_2$	>C=CH-$(CH_2)_n$-CH=C<
COH	COONO	
COOH	$COONO_2$	
COOH	CH_2ONO_2	
2. Others: CH_2OH-CH=C(COOH)-CHO CH_2OH-CH_2-CH=C(COOH)-CHO CHO-CH=CH-CH(CH_3)CHO CH_2OH-CH=CH=CH_b-C(CH_3)CHO $C_5H_8O_3$ isomers Nitrocresols $C_6H_6O_2$ isomers[b]		2. Not known; possibly from aromatic ring cleavage
Aromatic monofunctional compounds:		
3. C_6H_5-$(CH_2)_n$-COOH (n = 0,1,2,3)		3. Alkenylbenzenes C_6H_5-$(CH_2)_n$-CH=CHR; also toluene for C_6H_5COOH
4. C_6H_5-CH_2OH C_6H_5CHO Hydroxynitrobenzyl alcohol		4. Toluene, styrene, other monoalkylbenzenes?
Terpene-derived oxygenates:		
5. Pinonic acid Pinic acid Norpinonic acid		5. α-Pinene
6. Isomers of pinonic acid:[b] $C_9H_{14}O_2$ isomers $C_{10}H_{14}O_3$ isomers $C_{10}H_{16}O_2$ isomers		6. Other terpenes?

[a] Compounds identified at West Covina, Calif., July 24, 1974

[b] Isomers not resolved by mass spectrometry

processes. Such meteorological influences obscure both the absolute and the relative effects of the emission-related factors to degrees that vary with geographical location and season. Therefore, for proper assessment of emission-related factors, it is essential that meteorological factors and their effects on the oxidant problem be well understood. Such understanding often depends on meteorological details that are far beyond the scope of this discussion, but some general climatic factors may be discussed to illustrate their impact on air quality.

ATMOSPHERIC MIXING

The diurnal urban emission pattern for oxidant-forming pollutants is fairly uniform from weekday to weekday. It is apparent, therefore, that variations in daily oxidant/ozone accumulation must be attributable largely to meteorological factors. Of these factors, atmospheric mixing has an extremely significant effect on oxidant formation.

The rate and extent of atmospheric mixing and diffusion depends on stability, wind speed, and topography. Temperature inversions are relatively stable layers that inhibit atmospheric diffusion. They are common at night in layers near the ground, but they may also occur at higher altitudes and at times other than at night. Also, several successive atmospheric layers with different degrees of stability are not unusual. The morning peak concentrations of oxidant/ozone precursors

TABLE 4-5. RELATIVE IMPORTANCE OF ALIPHATIC AND AROMATIC PRECURSORS[a]

Gas-phase hydrocarbon precursors		Secondary organic aerosols				
		$X-(CH_2)_n-Y$		Concentration,[b] μg/m³		
		X	Y	n=3	n=4	n=5
		COOH	CH_2OH	2.18	3.40	0.65
		COOH	COH	1.39	2.59	0.82
		COOH	COOH	1.35	0.78	0.15
$(CH_2)_n\begin{smallmatrix}CH\\CH\end{smallmatrix}$		COOH	CH_2ONO	1.01	0.40	0.27
	or	COH	CH_2ONO_2	—	—	—
		COH	CH_2OH	0.31	0.40	0.13
		COH	COH	0.30	0.24	—
and/or	or	COOH	COONO	0.14	0.24	—
		COH	$COONO_2$	—	—	—
		COH	COONO	1.01	0.14	—
		COOH	$COONO_2$	—	—	—
		COOH	CH_2ONO_2	0.12	0.15	—
$>C=CH-(CH_2)_n-CH=C<$		Total		7.81	8.34	2.02
		$COOH-CH_2-COOH$		0.15	—	—
		$COOH-(CH_2)_2-COOH$		0.57	—	—
		Total difunctional compounds: 18.89				
$C_6H_5-CH=CHR$		$C_6H_5-(CH_2)_n-COOH$			n=0:0.38	
					n-1:0.41	
					n-2:0.52	
					n-3:0.03	
		Total from aromatics:			1.34	

[a] Of aerosols in Pasadena, Calif., Sept. 22, 1972; sampling period, 7:30 a.m. to 12:35 p.m.

[b] The same response factor (that of adipic acid) was used for all difunctional compounds.

often result from emissions from ground sources discharged into a stable atmosphere below an inversion layer. Typically, these low-level inversions begin to lift and provide a greater mixing height as the sun rises and heats the earth's surface. Thus mixing height (that is, the height above surface through which relatively vigorous vertical mixing occurs) varies during the day and with the season. Figure 4-3[65] shows average mixing heights in summer (usually the season of highest photochemical oxidant concentrations) that occur a few hours after sunrise. As surface heating continues, the low-level inversion is usually eliminated completely and, as depicted in Figure 4-4,[65] the mixing height increases to a maximum in the afternoon. Notice that these average afternoon mixing heights range from 600 m (1970 ft) along the California coast to more than 4000 m (13,000 ft) within the southern Rocky Mountain area. Over inland areas, the afternoon mixing heights are greatest in summer and least in winter (about half of the summer values). However, seasonal variations in coastal regions are usually small. To a large extent, the height of the afternoon mixing layer has a significant impact on the day's accumulation of oxidant/ozone.

The rate at which relatively unpolluted air moves into a region is related to wind speed. Wind speed and direction are highly variable in time and space (Figures 4-5 and 4-6).[65] The depicted wind speeds are averages of those speeds within the respective mixing layers. These averages tend to smooth out the greater variations in space and time that would occur if only surface winds had been considered. During summer mornings, the slowest average speeds are less than 2 m sec^{-1} and occur in the Far West. However, in the morning, average speeds of 3 m sec^{-1} or less occur over most of California and Oregon as well as part of Nevada; in the East they occur over the Central Appalachians and Mississippi. On summer afternoons, the average wind speed pattern is similar to that in the morning, except that the average speeds in the afternoon tend to be 1 to 2 m sec^{-1} higher than in the morning. This diurnal variation is generally true for all seasons.

Using daily values of morning and afternoon mixing height and wind speed, the occurrence of limited mixing episodes was determined for 62 weather stations in the contiguous United States.[65] The most limiting conditions were mixing heights of 500 m (1640 ft) or less, wind speeds of 2

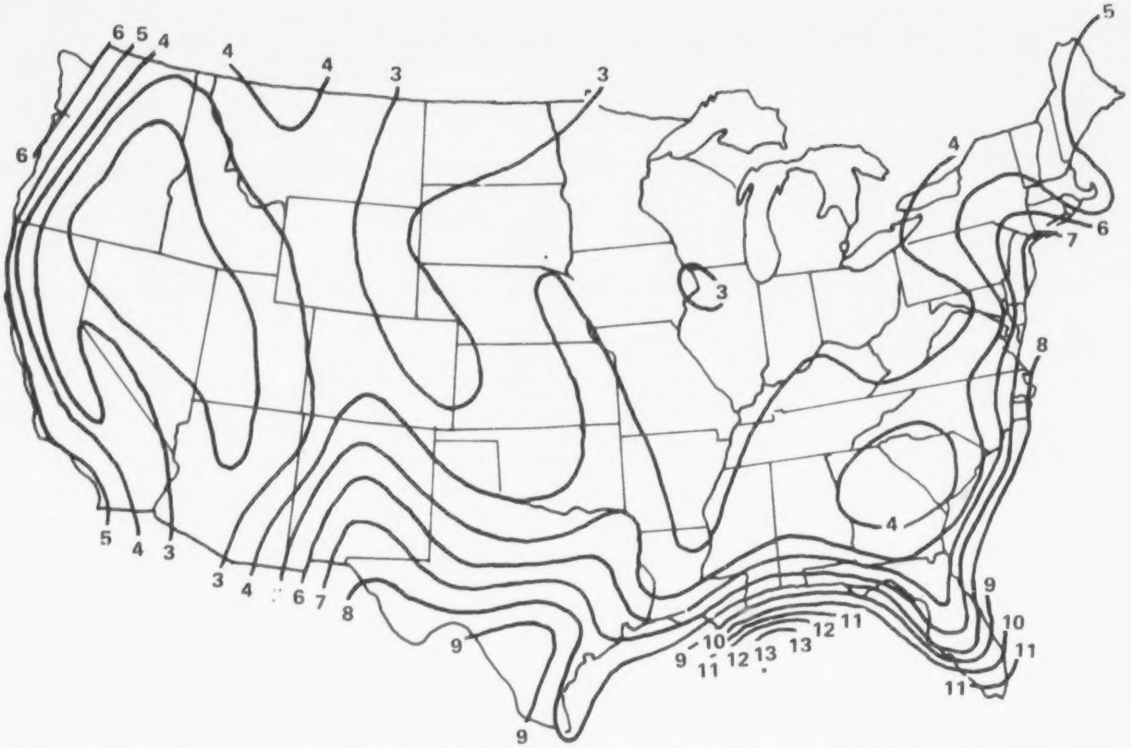

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Figure 4-3. Isopleths (m × 10²) of mean summer morning mixing heights.[65]

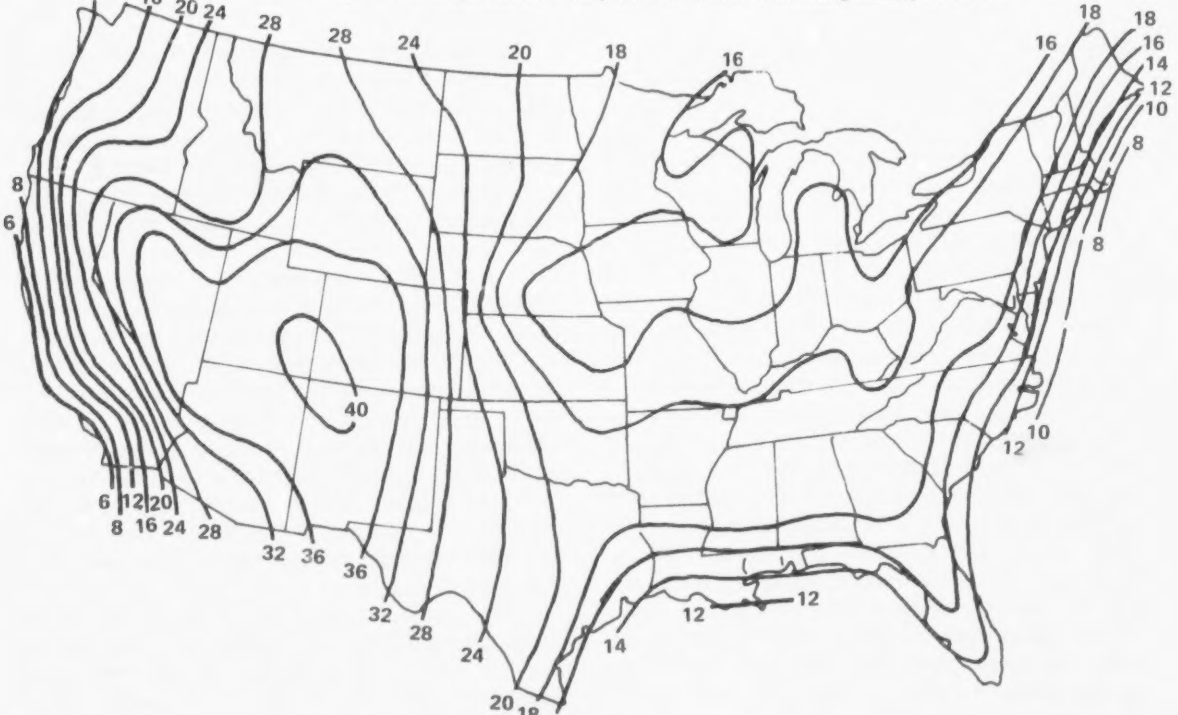
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Figure 4-4. Isopleths (m × 10²) of mean summer afternoon mixing heights.[65]

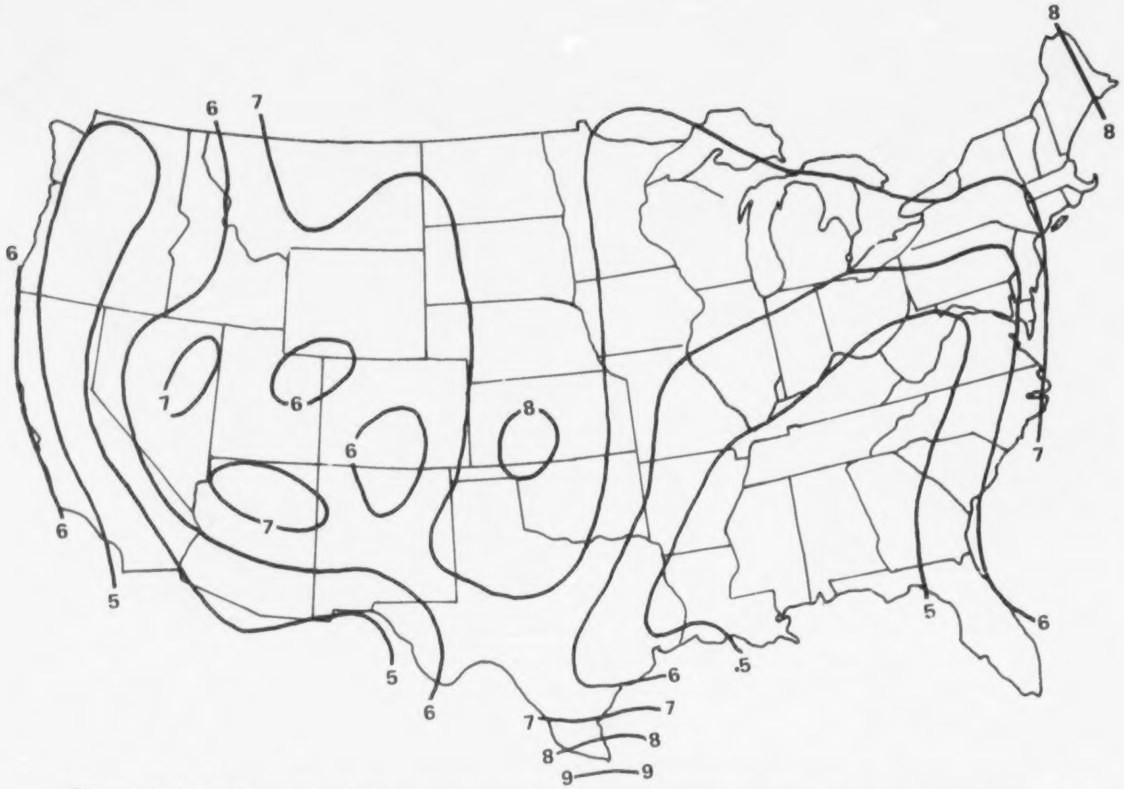
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Figure 4-5. Isopleths (m sec⁻¹) of mean summer wind speed averaged through the afternoon mixing layer.[65]

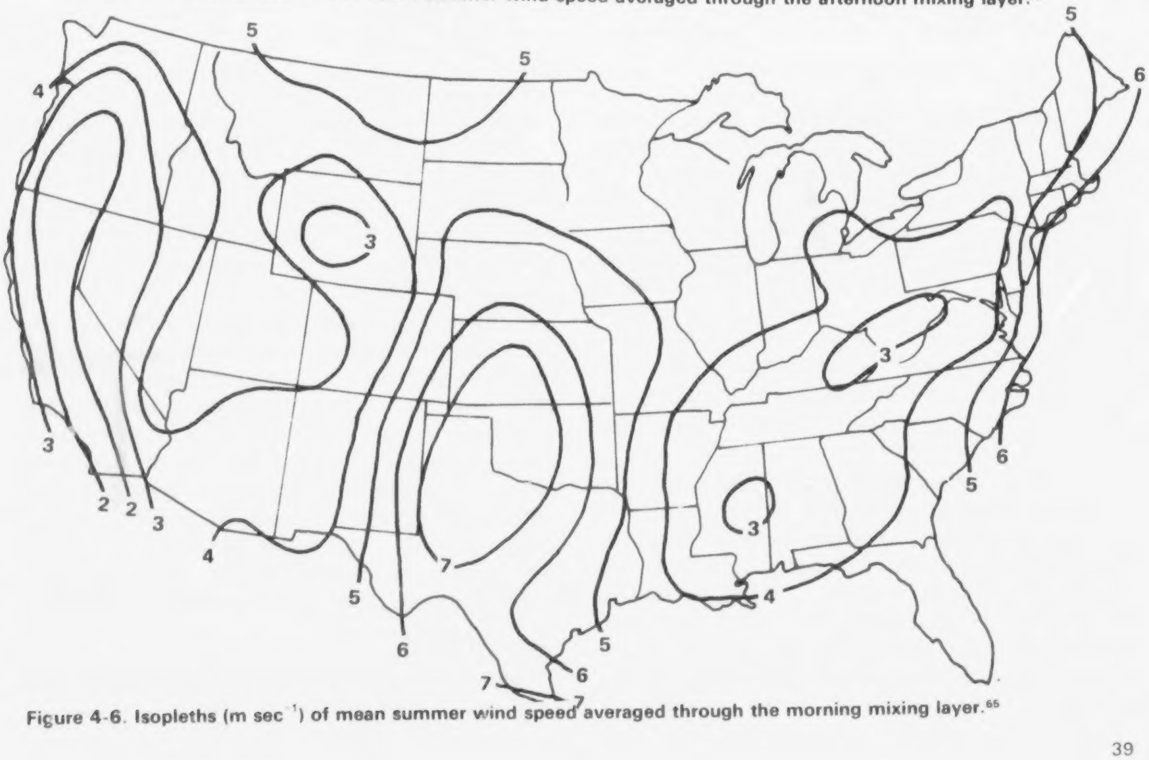
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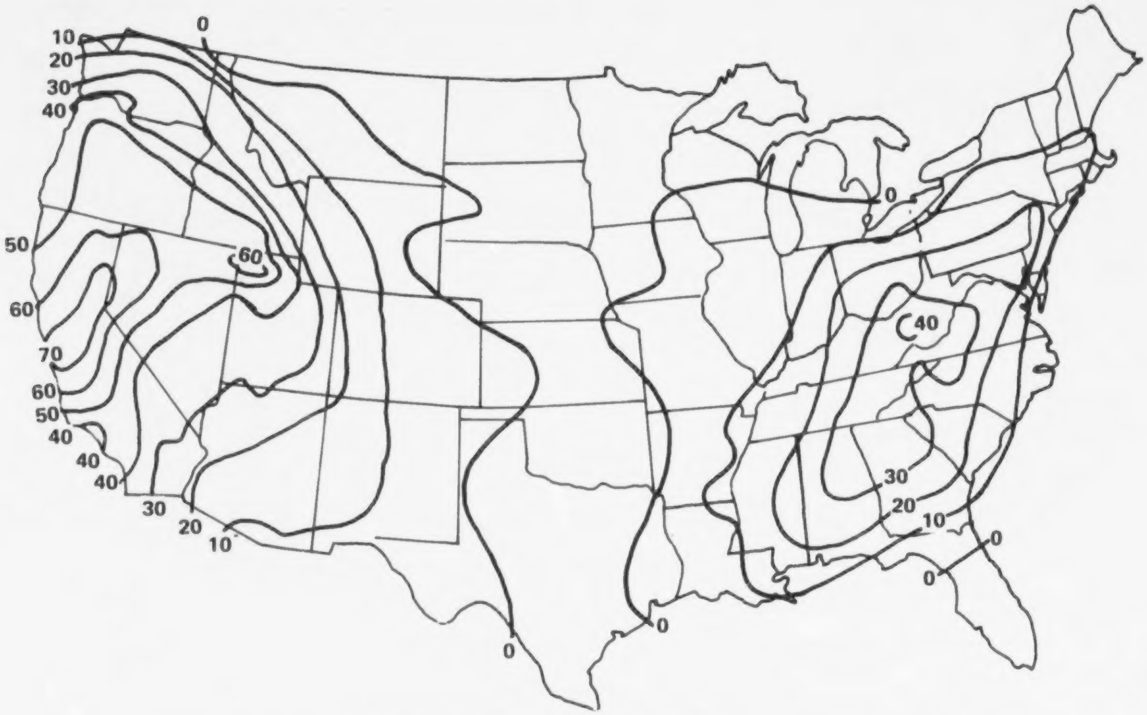
Figure 4-6. Isopleths (m sec⁻¹) of mean summer wind speed averaged through the morning mixing layer.[65]



DATA BASED ON FORECASTS ISSUED:
1 AUGUST 1960 TO 3 APRIL 1970 FOR EASTERN PART OF THE UNITED STATES
1 OCTOBER 1963 TO 3 APRIL 1970 FOR WESTERN PART OF THE UNITED STATES

Figure 4-7. Isopleths of total number of forecast days of high meteorological potential for air pollution.[65]

m sec^{-1} or less, and no precipitation during at least 5 consecutive days. Only six such epidoses occurred with a total of 39 episode-days at two stations (Medford, Oregon, and Lander, Wyoming) over a 5-year period. On the other hand, at least one episode of 2 days' duration, with mixing heights of 1000 m (3300 ft) or less and wind speeds of 6 m sec^{-1} or less, occurred at each of the 62 stations. In general, limited mixing episodes are most common in the Far West and within the Rocky Mountain region, least common over the Plains States, and of intermediate frequency east of the Mississippi (Figure 4-7).[65]

Korshover[76] compiled statistics on stagnation episodes occurring during the various months of the year east of the Mississippi. Such statistics are illustrated by graphs in Figures 4-8 and 4-9. Korshover's data, however, suffer from two limitations. First, stagnation episodes were determined indirectly, using sea level pressure gradient data, rather than the more direct indices of mixing height and wind speed; second, the requisite pressure gradient data could be obtained only for the relatively low and flat terrain east of the Rocky Mountains. A more detailed discussion of the Korshover statistics is included in the chapter dealing with the natural sources of oxidant/ozone.

In conclusion, the atmospheric mixing parameters have a strong effect on both the accumulation of oxidant-precursor pollutants and the photochemical oxidant-forming process. The effect on the latter process, however, although certain to exist, has only recently been studied directly and comprehensively;[73,118] changes in these mixing parameters cannot be simulated easily in smog chambers. Nevertheless, the information presented here is useful in that it suggests the areas and seasons in which atmospheric mixing conditions are relatively more conducive to oxidant formation and accumulation.

SUNLIGHT

The significance of sunlight is related to the intensity of sunlight and its spectral distribution,

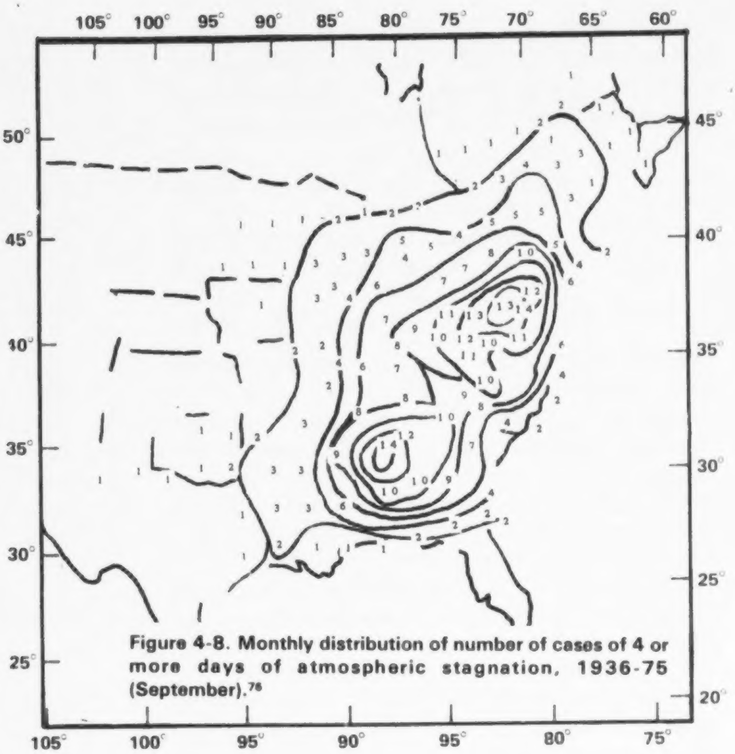

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Figure 4-8. Monthly distribution of number of cases of 4 or more days of atmospheric stagnation, 1936-75 (September).[76]

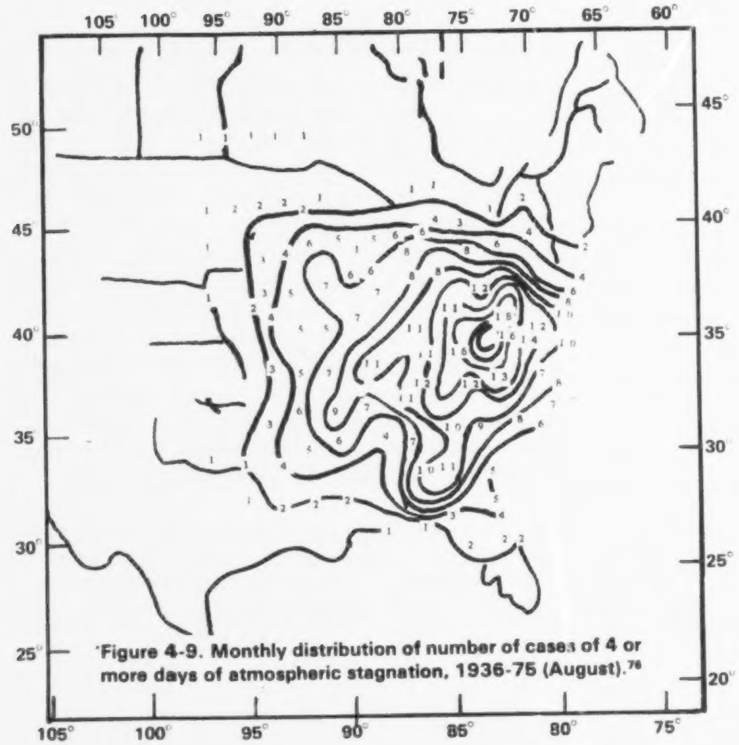
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Figure 4-9. Monthly distribution of number of cases of 4 or more days of atmospheric stagnation, 1936-75 (August).[76]

both of which have direct effects on the specific chemical reaction steps that initiate and sustain oxidant formation. Steps of varying significance include the following (and possibly others also):

$$NO_2 + h\nu \rightarrow O + NO \quad (4\text{-}19)$$
$$HONO + h\nu \rightarrow OH + NO \quad (4\text{-}20)$$
$$H_2CO + h\nu \rightarrow H + HCO \quad (4\text{-}21)$$
$$RCHO + h\nu \rightarrow CH_3 + HCO \quad (4\text{-}22)$$
$$O_3 + h\nu \rightarrow O\,(^1D) + O_2 \quad (4\text{-}23)$$
$$H_2O_2 + h\nu \rightarrow 2OH \quad (4\text{-}24)$$
$$HNO_3 + h\nu \rightarrow OH + NO_2 \quad (4\text{-}25)$$

Whereas the effect of sunlight intensity is direct and amply demonstrated,[78] the effect of wavelength distribution on the overall oxidant formation process is a subtle one. Experimental studies have shown the photolysis of aldehydes to be strongly dependent on radiation wavelength in the near-UV region.[28] Since aldehydes are major products in the atmospheric photo-oxidation of HC-NO_x mixtures, it is inferred that the radiation wavelength should have an effect on the overall photo-oxidation process. This inference was directly verified, at least for the propylene/NO_x and *n*-butane/NO_x chemical systems, in recent smog chamber studies.[70] In the ambient atmosphere, some variation in the wavelength distribution of sunlight does occur as a result of variations in stratospheric ozone, ambient aerosol,[131] and cloud cover. Obviously, such variation should be recognized and properly considered in conducting and interpreting outdoor experimentation. Beyond this consequence, however, the wavelength distribution factor has no other practical significance.

Sunlight intensity varies with season and geographical latitude, as shown in Figure 4-10.[78] The latitude effect is strong, but only during the winter months. During summer, throughout the contiguous United States, the maximum light intensity is fairly constant, and only the duration of the solar day varies to a small degree with latitude. Some variations in light intensity also occur with longitude during the summer months, with the highest intensities occurring in the western United States.

Absolute levels of sunlight intensities were calculated by Leighton nearly two decades ago.[78] Since then, several investigators have recalculated actinic fluxes and measured intensities by more direct methods. Thus recent measurements and recomputations by EPA investigators gave results somewhat different from those reported by Leighton. These differences are summarized in Table 4-6.[100] The recomputed values are lower than Leighton's in the 295- to 395-nm wavelength interval, slightly higher in the 395- to 450-nm interval, and considerably higher in the 450- to 700-nm interval. The same EPA investigators expressed their recomputed light intensities also in terms of values for the NO_2 photodissociation constant, k_1 (min^{-1}).[101] Resulting values are listed in Table 4-7. These values are higher than the earlier values used in laboratory simulations of polluted atmospheres. Jackson et al.,[69] Zafonte,[147] and Harvey et al.[56] measured k_1 directly; their values also were higher than those based on Leighton's calculations or on data obtained by standard radiometric techniques.

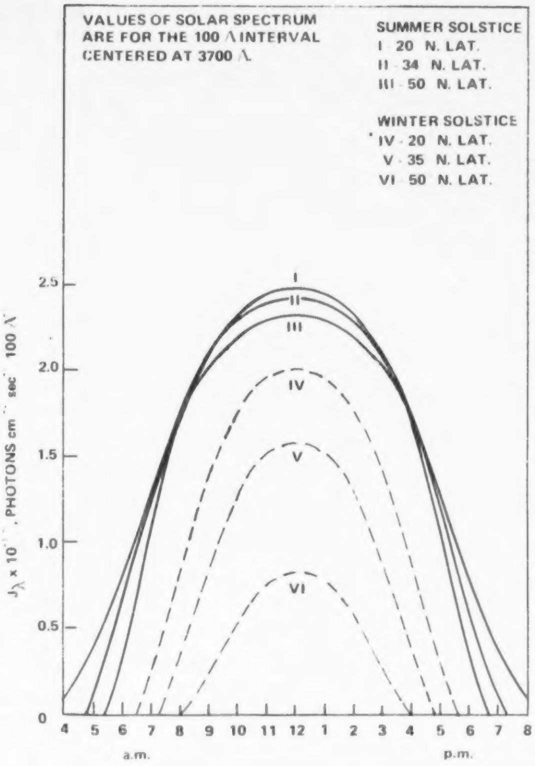

Figure 4-10. Diurnal variations in actinic irradiance.[78]

TABLE 4-6. PERCENTAGE DIFFERENCE BETWEEN THE PETERSON[100] AND THE LEIGHTON VALUES AT THE EARTH'S SURFACE OVER SELECTED WAVELENGTH INTERVALS AND SOLAR ZENITH ANGLES

Wavelength,	Zenith angle (°)			
nm	0	20	40	60
295-395	-6.1	-6.4	-5.5	-4.3
395-450	+2.4	+2.1	+3.4	+5.9
450-700	+18.8	+18.4	+17.0	+15.7

TABLE 4-7. CALCULATED VALUES OF NO_2 PHOTODISSOCIATION RATE CONSTANT[a] AT THE EARTH'S SURFACE AND PERCENTAGE INCREASE OF THE RATE CONSTANT FROM THE SURFACE TO VARIOUS HEIGHTS[101]

	Zenith angle (°)									
Height	0	10	20	30	40	50	60	70	78	86
Surface	0.579	0.574	0.560	0.535	0.496	0.432	0.352	0.231	0.114	0.025
0.15 km	6.0	6.1	6.4	6.9	7.7	8.9	11.1	14.3	16.7	8.0
0.36 km	11.4	11.5	12.1	13.1	14.5	16.9	21.0	27.7	34.2	20.0
0.64 km	16.6	16.9	17.7	19.1	21.2	24.4	30.4	40.7	52.6	35.0
0.98 km	21.4	22.0	22.9	24.7	27.2	31.5	39.2	52.3	70.2	48.0
1.84 km	29.9	30.3	31.6	34.0	37.7	43.4	53.7	73.2	101.0	72.0
2.91 km	36.4	37.1	35.8	41.7	46.2	53.7	66.6	91.3	132.0	100.0

[a] In units of min^{-1}.

The light intensity factor has been studied in the laboratory both with respect to its effect on individual photolytic reaction steps and with respect to its effect on the overall process of oxidant formation.[94] All of the early studies, however, employed constant light intensity conditions, in contrast to the diurnally varying intensity in the ambient atmosphere. Only within the last 3 years has the diurnal variation of light intensity been recognized and studied as a factor. Such studies have shown this factor to have a varying (with initial reactant concentration conditions), rather unpredictable, but somewhat significant effect.[72]

TEMPERATURE AND RELATIVE HUMIDITY

Effects of temperature and relative humidity on photochemical oxidant formation were suggested by early laboratory studies discussed in the predecessor of this criteria document.[92] Additional laboratory and field studies in more recent years verified a significant, positive temperature effect.[10,73] Thus outdoor smog chamber studies of synthetic HC-NO_x mixtures showed that in winter, with maximum daily temperatures at 50° to 60°F (10.0° to 15.6°C), oxidant yields were lower than those in the summer (80° to 90°F, or 26.7° to 32.2°C) by 70 to 90 percent, depending on reactant composition.[73] Also, an RTI analysis of field data showed (1) good correlation between daily maximum 1-hr oxidant levels and daily maximum temperature; and (2) no oxidant concentrations greater than 0.08 ppm on days when temperatures were below 62°F (16.7°C). The evidence[10,73] suggests that below about 55° to 60°F (13° to 16°C), photochemical oxidant concentrations are not likely to exceed the 0.08 ppm standard. Also, information on the temperature factor indicates that in the summer, temperature conditions are conducive to ambient oxidant formation throughout the United States as far north as Alaska.

Unlike the temperature factor, the effect of relative humidity remains somewhat uncertain. Reveiws by Quon and Wadden[103] and by Altshuller and Bufalini[4] have pointed out inconsistencies in the available evidence. Model simulations of irradiated HC-NO_x predict only a small humidity effect;[94] however, these predictions have not been verified by consistent experimental evidence.

TRANSPORT PHENOMENA

Relative contributions of natural and anthropogenic sources in a given region or area are a major question relevant to the ambient oxidant/ozone problem. This question has not been answered unequivocally and quantitatively chiefly because anthropogenic pollutant transport makes it difficult to assess the strength of the natural sources. The question has been answered qualitatively. The consensus is that oxidant/ozone and/or oxidant precursor transport does occur and contributes to oxidant/ozone buildup in areas far from the sources. The magnitude of this contribution and the magnitude of the contributions from the stratosphere and from other natural sources have been viewed as unresolved issues.[43]

Aside from its connection with the natural-versus-anthropogenic-sources question, the phenomenon of oxidant transport is important for another reason. The phenomena of urban oxidant/ozone plume formation and movement, rural oxidant/ozone occurrence (at problem levels), Sunday-weekday effect, and nighttime oxidant/ozone occurrence were previously either unnoticed or thought to be odd. They are now believed to be true manifestations of an extremely complex emission/pollutant dispersion process. Such complexities may be introduced by horizontal and/or vertical transport of oxidant/ozone and/or of precursor mixtures for long distances without excessive dilution.

Consequences of pollutant transport are discussed here. Several studies concerned with oxidant transport have been conducted in the last 5 years. Such studies have resulted in a wealth of information. This information and its implications are presented and discussed in the remainder of this section.

The first item of interest is evidence on the occurrence and range of oxidant transport. It should be clarified that the term "oxidant transport" is commonly used to refer to transport of either oxidant/ozone or oxidant precursors. The distinction between oxidant/ozone and oxidant precursors should be kept in mind, as it is essential to the understanding of the nature, mechanisms, and implications of the oxidant pollution problem.

Oxidant transport has been recognized to occur on three scales in terms of geographical distance or area subjected to the transport effects. These scales are:

1. The urban-scale transport, as a result of which the peak oxidant concentrations develop in the suburbs some miles downwind from the city core area where the oxidant/ozone and their precursors originated;
2. The mesoscale transport that encompasses land- and sea-breeze circulation, and the formation of urban oxidant plumes that create oxidant problems as far as 100 miles or more downwind from the source city; and
3. The synoptic-scale transport, a much longer and broader range of transport associated with high pressure systems.

Urban-scale transport has been observed in Los Angeles,[134] New York, Houston, Phoenix, and several other urban centers.[3,27] All of the evidence available points to the conclusion that conditions at the center of source-intensive areas are not the most conducive conditions for oxidant accumulation, mainly because of the strong scavenging effect of oxidant precursors, especially nitric oxide. At higher elevations or at horizontal distances downwind from the sources, where the precursor scavenging effect is less important, oxidant concentrations are, in general, greater, and their levels are determined primarily by the intensity of the photochemical activity and by ambient dilution. Measurements in several cities during days with various wind speeds showed the peak oxidant concentrations to occur at distances 8 to 136 km (5 to 85 miles) downwind from the city center.[87] During days of stagnation when the highest levels of oxidant/ozone occur, this peak-concentration distance was estimated by EPA to be between 15 and 25 km (9 and 16 miles),[53] and more recently, to be 15 to 30 km (9 to 19 miles).

This urban-scale transport of oxidant has two important implications. First, it suggests an obvious guideline to be used in siting stations for oxidant/ozone monitoring. The second implication, suggested by experimental data on the oxidant-to-precursor dependencies,[42] is that urban-scale transport may obscure the real impact of emission control on oxidant air quality. This latter implication may be clarified as follows: small or even moderate decreases in emission rates for HC and NO_x are expected to affect mainly the time at which the oxidant concentration will reach the day's peak levels; the effects on the peak level itself are of smaller magnitude. This time lag effect, in turn, translates into an increase in the distance from the source area where peak oxidant concentrations develop (i.e., the location of the peak concentration relative to the location of the core of the source area). Thus, for example, the actual effect of a modest HC emission reduction will be to shift the peak oxidant concentration farther downwind, rather than to effect a proportional reduction locally. Therefore, the impact of emission control will appear to be either beneficial or detrimental, depending on the location of the oxidant monitoring station.

Evidence on occurrence of mesoscale and synoptic-scale transport has been obtained in numerous extensive and conclusive studies. These studies have been reported individually[10-12,25,26,28,30,38,39,44,51,61,66,68,85,86,115,116,129,130,136,138,139,-145] and collectively in reviews.[85,97,120] The evidence shows that many cities produce urban oxidant plumes that cause elevated oxidant concentrations in downwind areas as far as 300 km (190 miles) or more from the source city.[12,25,61,74,128,136,143] Transport associated with synoptic-scale high pressure systems, unlike such mesoscale transport, is not characterized by well defined urban oxidant plumes and can extend to distances several times greater than 300 km (190 miles).[38,39,97,115,116,120,136,145] In either case, the significant implication is that urban emissions may be creating oxidant problems not only within and near their source areas, but also in other downwind urban areas, as well as oxidant problems in rural and even in remote areas. It should be stressed that the evidence on horizontal transport of oxidant/ozone suggests but does not prove that most of the oxidant observed in rural and remote areas is transported from the cities. Also, horizontal transport of oxidant over long distances can only occur in air layers aloft; ozone at ground level is rapidly destroyed on surfaces and in reactions with NO and NC.[23,28,39,74,118,136] For oxidant/ozone to reach the surface, there must be vertical air movement, a movement that may also bring to the surface ozone from the stratosphere or from other natural sources. In general, because of the multiplicity of the sources that potentially contribute to surface oxidant/ozone buildup, the rural oxidant/ozone problem cannot automatically be attributed entirely to urban oxidant/ozone transport. The indications are that direct urban transport is a relatively important source of oxidants in many locales but not necessarily everywhere.

Because of their elevated ambient oxidant/ozone levels, the State of California, the Gulf Coast and Texas, the upper Midwest, and the Northeast are the areas of the United States that have been studied most. The explanation for oxidant/ozone levels in California is relatively simple: Ozone levels in that area can be explained by local photochemistry and oxidant/ozone transport; nonanthropogenic explanations are not required. The most striking and clearly explained oxidant transport case reported for that area is the one involving overnight transport over water of Los Angeles air containing as much as 590 $\mu g/m^3$ (0.3 ppm) oxidant to San Diego.[12,120]

Unlike California, the situation in the Gulf Coast/ Texas area is considerably more complex and requires more careful examination. Early studies in Texas led investigators to conclude that widespread ozone concentrations were not of anthropogenic origin. The basis of that early conclusion was the observation that ozone was frequently high when there were onshore winds at coastal stations.[120] However, recent studies by Price[102] and by Decker et al.[38] at the Research Triangle Institute (RTI) concluded that natural tropospheric ozone was not a factor in the high ozone events in Texas. The RTI study, in particular, involved extensive aerial and ground measurements of ozone, HC, and NO_x, and wind trajectory analysis. Results from that study showed the high ozone concentrations over the Gulf to be associated with recent passage of the air stream over land areas and often over source-intensive areas. This finding, similar to that by Bell for the Los Angeles/San Diego case,[12] explains more rationally the observations made in the early studies of Texas and implies that the Texas ozone problem is at least partly of anthropogenic origin. The RTI study also indicated an association of high ozone concentration with the passage of high pressure systems, an association which had been recognized for years and which was more firmly established in the midwestern part of the United States.

Transport of ozone for an unusually long distance was reported in an episode of widespread haze over southern Florida from May 21 to 27, 1972.[123] The initial report concerned metropolitan Dade County and was supplemented by concurrent data obtained at the site of a proposed jetport 65 km (41 miles) west of Miami. At this rural site, the ozone concentration equaled or exceeded 160 $\mu g/m^3$ (0.08 ppm) on 5 of those days, and the air quality standard was exceeded for 14 consecutive hr on both May 22 and 23. The analysis of the transporting trajectory that brought the haze and accompanying ozone was verified by the EPA Meteorological Laboratory. The source region appeared to be the industrial area among the states south of the Great Lakes, making a transport distance of over 1000 miles (1600 km), of which 400 miles (640 km) were over the Gulf of Mexico. Both the haze and the ozone of its precursors may have been augmented along the route.[46]

Most of the oxidant transport studies in the upper Midwest dealt with the role of high pressure meteorology in wide area oxidant problems. The connection between stagnating anticyclonic conditions and high oxidant concentrations over large areas was studied by several investigators and is now believed to be relatively well understood.[38,39,115,116,120,129,130,136,138,139,145] This belief is based on (1) data from direct monitoring with an aircraft of the ozone concentrations in the path of a moving high pressure system, and (2) presence of statistically supported correlation between high pressure systems and elevated ozone concentrations during days with such conditions. This correlation has been verified by numerous studies. The reverse has not been established; that is, elevated ozone levels do not occur only during stagnating anticyclonic episodes.

Within the moving and clockwise rotating anticyclone, the highest ozone concentrations are observed in the trailing or western portion of the cell where the air parcels have the longest residence time in the anticyclonic regime. RTI investigators made some detailed model calculations to show that air parcels initially in the northeast quadrant of the cell had a potential residence time as long as 6 days, and that air parcels on the western side of the circulation pattern had been in the cell the longest time. This latter conclusion is in general agreement with ozone measurements in source areas such as have been described by Westberg et al.[138] Under conditions of the high intensity sunlight and stagnation or undiluted flow that typically prevail within the anticyclone, oxidant/ozone formation is expected to be accelerated and depends on the emission density within the area covered by the high pressure system. This latter dependence seems to be manifested by the data obtained in the paths of high pressure systems moving from less to more densely populated areas.[38]

A significant percentage of the high oxidant/ozone episodes observed in the United States is associated with passage of high pressure systems. Statistics of high pressure episode occurrence have been obtained by Korshover[76] for the eastern part of the United States for all months of the year. The study could not be extended in the western United States because the extreme terrain irregularities result in unrepresented pressure distributions. Such statistics for the months of August and September are shown by the plots in Figures 4-8 and 4-9.

Oxidant transport in the Northeast received considerable attention because high oxidant/ozone concentrations have been measured almost routinely in rural and distant suburban areas for years. Careful analysis by Bell Laboratories and other investigators of aerometric data obtained in northern New Jersey, eastern New York, Connecticut, and Massachusetts provided strong evidence of extensive mesoscale transport, especially from the New York City/New Jersey area to the Northeast into Connecticut and Massachusetts.[120,146] Such transport was characterized (1) by low oxidant/ozone levels within urban core areas because of scavenging by precursors, (2) by appearance of highest oxidant/ozone concentrations downwind from the main urban center at a distance equivalent to 1 to 2 hr of midday wind travel, and (3) by shifting of peak oxidant/ozone concentrations with time and distance, reflecting the regional movement of the main urban area plume along the wind trajectory. The results from Bell Laboratories seem to be in agreement with subsequent studies by other groups. Interestingly, the evidence descriptive of the Northeast situation suggests that weather patterns seem to play a minor role in establishing oxidant/ozone levels in that part of the United States. For example, oxidant/ozone concentrations seem to be less sensitive to the location or intensity of an anticyclonic system.[120] This could be a result of the high population density in that area and the relative dominance of urban plumes over the nonanthropogenic background levels in the air mass. Finally, local circulations in the surface layers were found to play a dominant role in the coastal areas in the Northeast.[120,139] Such circulation systems, subjecting areas to next-day effects from their own emissions, were also observed in the Gulf Coast[38] and Midwest[86] areas but were not as dominant.

The evidence on transport has had another important impact on the understanding of the oxidant/ozone problem. It provided one plausible explanation to some phenomena previously thought to be paradoxical (e.g., the occurrence of elevated oxidant/ozone concentrations in rural areas and at night[115] and the Sunday-weekday effect[13,24,45,80,135]). Thus downward intrusion, from layers aloft, of transported anthropogenic oxidant/ozone explains convincingly the occurrence of elevated oxidant/ozone concentrations in rural areas and during the night in the summer-fall months. The Sunday-weekday effect could also be explained in part as resulting from anthropogenic oxidant/ozone present in layers aloft. Although these explanations have not been supported by documentation in all cases, they at least have served to clarify these phenomena and to increase confidence in the current knowledge and understanding of photochemical processes.

Identification and understanding of oxidant/ozone transport represents a significant advance in understanding photochemical pollution. However, it has also raised several questions that need to be addressed. For instance, the impact of pollutant transport on the oxidant concentration observed in a locality cannot be quantified at this time.[43] The difficulty here is not merely the absence of sufficient data (e.g., data on pollutant composition in transported, aged air masses), but also the absence of valid investigative methods. Clearly the need is for both laboratory and field studies that would provide a better understanding of the quantitative aspects of the interaction of transported, aged pollutant mixtures with locally emitted fresh pollutants. More specifically, a method should be developed for estimating the proportion of local oxidant/ozone concentrations resulting from pollutant transport.[43] Evidence must also be obtained on the oxidant-to-precursor dependencies for transported oxidant/ozone.[43]

NATURAL SOURCES OF OXIDANTS

Introduction

The occurrence of ozone in remote areas and during the winter months constitutes strong evidence that natural sources of ozone must exist, providing an atmospheric background on which the anthropogenically generated ozone is superimposed. Promulgation of the 160-$\mu g/m^3$ (0.08-ppm) National Ambient Air Quality Standard for ozone raised considerable interest in the quantity of emissions from natural sources because of the possibility that the natural sources alone could make it impossible to achieve the air quality standard. Additional interest was raised by the discovery that a widespread ozone problem existed in the rural areas, a problem that could conceivably have natural causes. The interest in these aspects of the question of natural sources is certainly justified, but it is also of a somewhat academic nature. More pragmatically, the question of the emission levels of natural sources needs to be answered to estimate more accurately the benefits to be derived from the reduction of anthropogenic emission.

Early research on natural ozone identified a number of sources, of which stratospheric ozone intrusion, electric discharges, and photochemistry of natural emissions appeared to be the ones with the highest and most probable contributions. It should be noted that much of the evidence in those early studies was indirect, based on reasoning by elimination; that is, observed ozone was attributed to natural sources when explanations for anthropogenic origins were eliminated, either because they could not be made or because they were not acceptable to the researchers. Subsequent studies, however, did produce some direct evidence, mostly on the stratospheric source and to a smaller degree on the photochemistry of natural emissions. The case for a significant contribution to background ozone from natural electric discharges has not been made convincingly. Thunderstorms have been observed on occasion to cause elevated short-term concentrations of ozone, but it is not clear whether such ozone is electrically generated or reflects stratospheric intrusion brought about by the thunderstorm disturbance.[46] Therefore the discussion in the following sections will focus only on stratospheric ozone and natural emission photochemistry sources for which there is a relatively greater amount of information available.

Stratospheric Ozone Intrusion

The magnitude of the tropospheric air quality problem caused by stratospheric ozone intrusion has long been an unresolved issue.[43] The specific questions at issue are whether such intrusions are sporadic, unpredictable incidents causing local, short-term ozone accumulations, or whether they are significantly extensive and predictable. Aside from the possibility that stratosphere-troposphere exchange contributes significantly to ground level ozone buildup, stratospheric ozone has also been proposed to have a reaction-trigger function that accelerates and enhances photochemical oxidant formation from HC-NO_x precursors.[57] Evidence interpreted to show accumulation of stratospheric ozone within the troposphere varies widely in type and degree of directness. In most cases, the evidence reported is based on ozone measurements in remote areas, measurements that do not necessarily represent stratospheric effects alone. Recently, Reiter,[111] Mohnen,[90] and Singh et al.[126] independently reviewed and critically examined the information available on the stratospheric ozone issue and offered perhaps the most reliable and definitive assessments of the stratospheric ozone problem to date. Based chiefly on their reviews and on other available literature, the case regarding stratospheric ozone intrusion appears to be as follows.

There are two types of evidence pertinent to the stratospheric ozone issue: (1) Evidence derived from analysis of global circulation patterns and, more specifically, from the quantification of the various known mechanisms or processes through which air is interchanged between the stratosphere and troposphere; and (2) evidence derived from analysis of data on tropospheric ozone concentration and concentration variation in remote areas. Estimates of stratospheric ozone concentrations at ground level may be drawn from both types of evidence, but with uncertainty. Estimates based on stratosphere-troposphere exchange data suffer mainly from uncertainties in the frequency and intensity of tropopause folding events and by the unquantified and hence inadequately considered ozone decay processes within the planetary boundary layer. Estimates based on tropospheric ozone measurements are also subject to uncertainty because of hidden influences from nonstratospheric ozone sources.

It is well established that air interchange between stratosphere and troposphere occurs via the following four mechanisms:[111] (1) mean meridional circulation, (2) large-scale eddy transports (jet streams), (3) seasonal adjustment of tropopause level, and (4) mesoscale and small-scale eddy transport. Each of these mechanisms is characterized by short-term, seasonal, and long-term fluctuations, and their impact in terms of surface concentration of stratospheric ozone varies with geographical latitude.

The mean meridional circulation (MMC) mechanism involves upward flux of tropospheric air mainly in the low latitudes and downward flux of stratospheric air in the middle latitudes. It varies somewhat in intensity from season to season, the most intensive period being in winter (December-February). Reiter[111] estimates the annual amount of stratospheric-MMC air mass transferred into the troposphere to be about 43 percent of the total mass of one stratospheric hemisphere, or about 1.84×10^{20} g of air. Reiter further estimates that the upper limit of average ozone concentration shortly above the tropopause is 0.05 $\mu g/g$. These estimates of mass exchange and the mean ozone concentration result in a calculated average ozone exchange of 9.22×10^{13} g of ozone due to the MMC process. Furthermore, assuming that the MMC ozone flux exchange is concentrated over half the area of one hemisphere, Reiter calculates a yearly mean ozone flux of 0.23×10^{-7} $g/m^2 \cdot sec$. During the winter period, however, when the strongest cross-tropopause fluxes occur, this ozone flux could be 0.4×10^{-7} $g/m^2 \cdot sec$. From this stratospheric mass flux (0.4×10^{-7} $g/m^2 \cdot sec$) and estimates of the mean tropospheric vertical velocity, Reiter calculated the mean background of stratospheric-MMC ozone in the springtime and in the midlatitudes to be 14.6 $\mu g/m^3$ (0.007 ppm). The yearly mean from Reiter's flux estimate would be about 8 $\mu g/m^3$ (0.004 ppm). Junge, as cited in Reiter,[111] using a somewhat higher flux value and a lower vertical velocity value, estimated such a mean background ozone concentration to be 35.6 $\mu g/m^3$ (0.017 ppm).[111]

Superimposed on the background concentration caused by the MMC process, there is an ozone increment of stratospheric origin caused by large-scale eddy mixing occurring in the jet stream regions through a tropopause folding (TF) process. The intensity of this process relative to the MMC process is somewhat uncertain; however, its impact per unit of ozone mass transported on the lower troposphere is unquestionably stronger and more localized. This is because relative to the MMC mechanism, stratospheric air during TF intrusion moves downward much faster and therefore is subject to much less tropospheric dispersion. Stratospheric ozone intrusions of the TF type occur mostly along the polar-front jet stream and yield, on the average, maximum stratospheric ozone accumulations at ground level in midlatitudes during March and April.

Using the studies of Danielsen, Mohnen[90] has examined the intensity of the TF process, as has Reiter,[111] using data from Mahlman. From case studies of TF events, Danielsen and Reiter estimated that the stratospheric air mass transported per TF event was 4 to 6×10^{17} g. From the average number of TF events observed during 1963-64, Reiter calculated the annual air mass transport to be equivalent to approximately 20 percent of the air mass in the northern stratospheric hemisphere. In contrast, Danielsen's estimate was 90 percent.

To estimate the impact of the TF-type stratosphere-troposphere interchange in terms of surface ozone concentration, it is necessary to know (a) the ozone concentration in the transported stratospheric air and (b) the (stratospheric) ozone decay process occurring within the troposphere. Indirect estimates of these have been made based on radioactive ^{90}Sr tracer methods and on isentropic trajectory analysis techniques. From measurements of the ^{90}Sr-to-ozone ratio within the lower stratosphere and from ^{90}Sr-related radioactivity data at ground level, Reiter re-estimated recently the 24-hr average surface concentration of stratospheric-TF ozone to be as high as 60 $\mu g/m^3$ (0.033 ppm) in 1963 and 120 $\mu g/m^3$ (0.066 ppm) in 1964.[113] Reiter's estimate must be considered an upper limit, since it assumes that the ^{90}Sr-to-ozone ratio is conserved during transport from the lower stratosphere to ground level. From use of isentropic trajectory analysis techniques, it was deduced that exceptionally strong TF-type intrusions occur only occasionally, but can cause surface ozone concentrations of 160 $\mu g/m^3$ (0.08 ppm) or higher. Such strong intrusions are expected to occur about once a year, usually in the southern and eastern United States. Reiter estimated the probability of such occurrence to be 0.2 percent, measured in days of observations on an annual basis at a given location.[111]

Direct evidence of the impact of stratospheric ozone on ground-level oxidant during TF events has been reported by Lamb.[77] He performed a detailed study of an incident that occurred in Santa Rosa, Calif., in which hourly averaged ground-level oxidant concentrations exceeded 160 μg/m^3 (0.08 ppm) for 5 consecutive hours just before dawn on November 19, 1972. The largest of the five hourly averaged concentrations was 450 μg/m^3 (0.25 ppm). All available evidence indicated that the ozone responsible for this episode originated in the stratosphere during a TF event off the south coast of Alaska 2 days earlier. Chatfield and Harrison[22] found a positive correlation between increases in ozone concentration in the remote Olympic Mountains of Washington and the passage of low pressure systems originating in the same region that the TF event responsible for the Santa Rosa episode occurred. Attmannspacher and Hartmannsgruber[9] have also reported ozone on an anomalous 1000-m (3300-ft) mountain peak in northern Germany attributable to stratospheric intrusion. On three different occasions during the winter of 1971, they observed fluctuations in ozone concentrations between 490 μg/m^3 (0.025 ppm) and 980 μg/m^3 (0.50 ppm) that lasted longer than 10 min. Each occurrence was during strong snow showers associated with passing cold fronts. (The Santa Rosa episode was initiated by a brief rain shower shortly after the passage of a cold front.) On three occasions, balloons were launched to measure ozone concentrations aloft, and in all three cases, a strong, secondary ozone concentration maximum was observed just above the tropopause.

Besides the MMC and TF processes, stratosphere-troposphere interchange occurs also the seasonal tropopause adjustment (STA) and the small-scale eddy transport (SSET) mechanisms. Of these, the STA mechanism has been estimated to cause a stratospheric air mass flux equivalent to 10 percent of the entire stratospheric hemisphere.[90,111] Such interchange occurs mainly in the same season and latitudes as the TF process. Therefore, its impact is included in the estimates made for the TF process. The SSET process is of much less importance and contributes to the tropospheric ozone problem only at the noise level.[90,111]

In conclusion, based on Reiter's estimates of stratosphere-troposphere interchange and on ^{90}Sr data, the annual average total of stratospheric ozone accumulation expected at groundlevel amounts to 20 to 30 μg/m^3 (0.01 to 0.015 ppm).[84,113] If one considers the fact that some ozone can be destroyed during transport to ground level, the preferred mean ozone concentration calculated by Reiter would be closer to the lower limit of 20 μg/m^3 (0.08 ppm). Occasional excursions to about 160 μg/m^3 (0.080 ppm) can be expected, however. Danielsen's estimates of stratospheric ozone flux are a factor of two to three higher than Reiter's estimates and would lead to correspondingly higher yearly mean ozone concentrations. Danielsen estimates an ozone flux rate of 8×10^{10} molecules/cm^2· sec. Singh et al.[127] suggests that this flux rate would be compatible with ground level ozone measurements at remote locations (yearly mean of 60 μg/m^3, or 0.030 ppm) if an ozone lifetime of about 4 months is assumed in the troposphere. The lifetime of ozone is uncertain, and all estimates of ground level ozone determined from stratospheric ozone intrusions suffer from this uncertainty. Both of Reiter's and Danielsen's estimates apply to situations where stratospheric ozone is brought to ground level without the assistance of precipitation-driven downdrafts. The case studies cited above by Lamb[77] and Attmannspacher and Hartmannsgruber[9] (and others) indicate that stratospheric ozone accompanying TF events can reach ground level in concentrations exceeding 390 μg/m^3 (0.20 ppm) when it is transported part of the way in downdrafts caused by precipitation. The frequency of this type of event is not yet known, but the mean ozone contribution resulting from such events is expected to be minimal.

Mohnen's estimate of the annual average stratospheric ozone at ground level is 40 to 70 μg/m^3 (0.022 to 0.035 ppm).[90] Such background levels vary with season and latitude because the stratospheric ozone reservoir also varies with season and because the various stratosphere-troposphere interchange mechanisms vary in intensity with season and latitude. Considering all these variables, there seems to be a consensus of opinion that at locations in the midlatitudes (e.g., the United States), the ground level ozone concentrations of stratospheric origin peak in winter and spring.

The preceding paragraphs dealt mostly with the evidence and conclusions obtained from analysis of global circulation patterns. As stated earlier in this section, relevant evidence was obtained also from analysis of tropospheric ozone concentration data in remote areas. Such evidence, although seemingly more valid as being more direct, should be examined carefully, for it is often distorted or obscured by hidden interfering factors. Examples of such factors are destruction of ozone on surfaces or in reaction with HC and NO, and photochemical formation of ozone.[106]

Mohnen[90] examined the evidence both on stratosphere-troposphere exchange and surface ozone concentration and concentration variation, but, unlike Reiter,[111] he elected to base his conclusions on stratospheric ozone intrusion mainly on tropospheric ozone data. Utilizing the analytical method introduced by Junge[90] and data from monitoring stations in remote areas presumably free of local or regional anthropogenic emissions, Mohnen concluded that a range of annual mean concentrations equal to 40 to 70 $\mu g/m^3$ (0.022 to 0.035 ppm) constitutes a representative tropospheric ozone level (of stratospheric origin) for 35° to 50° N latitudes. Mohnen also concluded that TF events may lead to ozone concentrations at ground level as high as 30 $\mu g/m^3$ (0.15 ppm) and lasting from 2 or 3 hr up to 1 to 3 days.

Singh et al.[126] examined aerometric data on ozone, HC, and NO_x obtained at 10 stations selected to be as remote as possible and with air quality records of at least 2 years' duration. Seven of these stations were in the western continental United States, one in Hawaii, one on the summit of Whiteface Mountain in New York, and one on the Zugspitze in West Germany at a 3000-m (9800-ft) elevation. The ozone variation patterns observed at most of these stations are exemplified by the patterns observed at the Quillayute, Washington, and Mauna Loa, Hawaii, stations (Figures 4-11 and 4-12). The striking features of these patterns are the maximum ozone concentrations observed in April and the 160-$\mu g/m^3$ (0.08-ppm) or higher ozone concentrations of such maxima. Considering the low sunlight intensity and temperature conditions prevailing in April at Quillayute, it can be deduced that these high ozone concentrations must be primarily of natural, stratospheric origin. Such a deduction is further supported by the lack of the familiar diurnal ozone variation pattern that characterizes ozone formation from local photochemistry. Significantly, this stratospheric ozone declines as summer months approach, indicating that the effects of stratospheric intrusion are at a low ebb during the smog season. Singh et al.[126] did observe elevated ozone concentrations during the summer months in some stations. Such maxima, however, were explained in terms of photochemical ozone, either locally produced or transported to the stations from upwind areas.

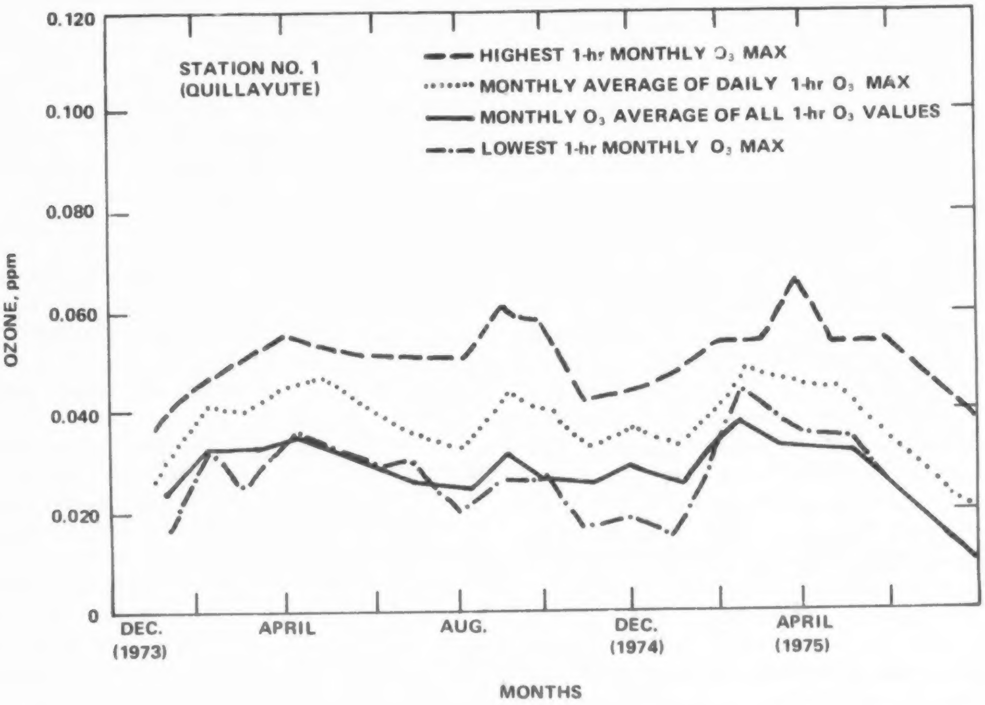

Figure 4-11. Long-term ozone variations at Quillayute.[126]

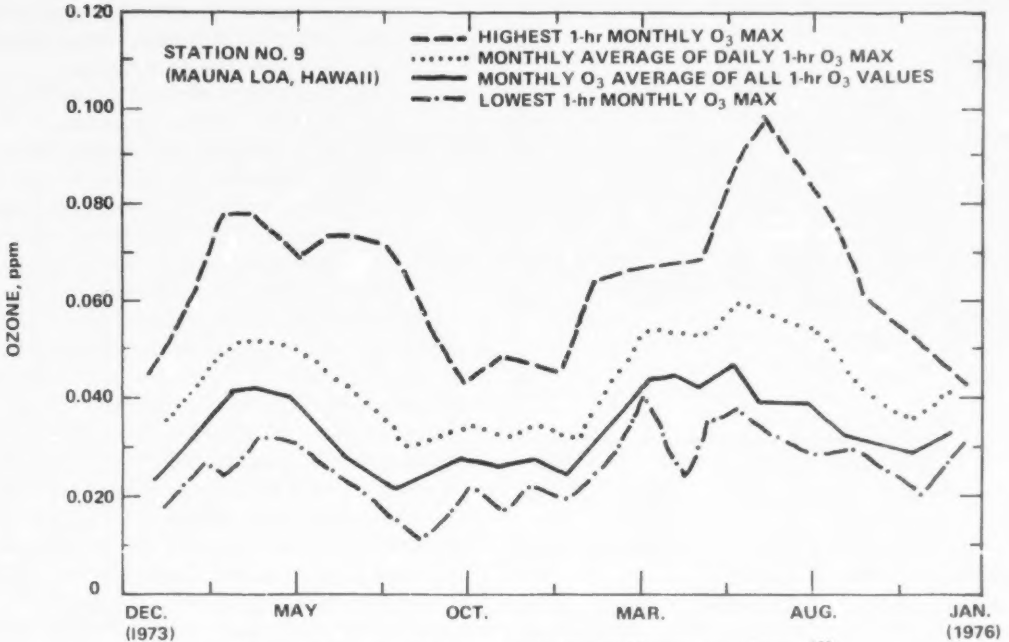

Figure 4-12. Monthly ozone variations at Mauna Loa, Hawaii.[126]

Data collected by Singh et al.[126,127] suggest yearly mean ozone levels of about 60 μg/m^3 (0.03 ppm) between latitudes 18° N and 48° N. The springtime mean values are found to be about 80 μg/m^3 (0.04 ppm), and the fall mean is about 40 μg/m^3 (0.02 ppm). The yearly 1-hr maximum ozone concentrations also show large year-to-year variation. For example, at Mauna Loa (Figure 4-12), the 1-hr ozone maximum of 160 μg/m^3 (0.08 ppm) was exceeded on 1.6 percent of the days in 1975 and on zero percent of the days in 1974.

The Whiteface Mountain data were examined also by Coffey et al.[28] along with data from other remote areas and from urban locations in New York State. The observations made by Coffey et al.[28] from these data are: (1) Ozone concentrations at Whiteface Mountain often exceed the 160-μg/m^3 (0.08-ppm) level; (2) a background ozone blanket covers the entire state; and (3) there is monthly variation of ozone with a strongly defined maximum in August (Figure 4-13). Presence of the ozone blanket suggests that the ozone source lies aloft and cannot be due to local photochemistry. However, occurrence of the peak concentrations in the summer (August), coupled with evidence on presence of manmade pollutants (halocarbons)[106] can only be interpreted to mean that the Whiteface Mountain data did reflect anthropogenic influences, a conclusion pointed out also by Reiter[111] and by Singh et al.[126] Thus transport, in paths aloft, of ozone and/or ozone precursors from upwind anthropogenic sources provides a more plausible explanation of the Whiteface Mountain data patterns, at least for 1973.

More recently, Husain et al.[67] obtained and analyzed ozone and ^{7}Be data in Whiteface Mountain during the smog season. In their analysis, these investigators used data on ^{7}Be and air trajectory analysis to establish stratospheric origin, and NO_x and aerosol concentrations and O_3-^{7}Be correlation patterns to verify absence of photochemical ozone. Interpretation of results suggested an upper 24-hr concentration limit of 75 μg/m^3 (0.037 ppm) stratospheric ozone at Whiteface Mountain during July 1975.

Measurements of ozone on the Zugspitze, West Germany, were analyzed both by Reiter[111] and by Singh et al.[126] Reiter's frequency distribution analysis of data obtained during August 1973 to October 1975 indicates that (a) 0.2 percent of the hourly concentrations exceeded the 160-μg/m^3 (0.08-ppm) level, and (b) relatively high concentrations occurred during summer rather than spring. The latter indication again suggests an anthropogenic-influences explanation. However, in this case, such an explanation alone is not

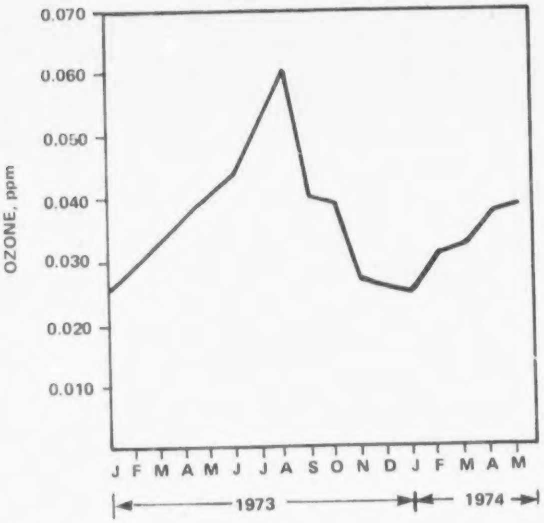

Figure 4-13. Average monthly ozone concentrations recorded at summit of Mount Whiteface.[128]

convincing because the Zugspitze station, at the altitude of 3000 m (9800 ft), is above the planetary boundary layer where anthropogenic pollutant transport occurs. Reiter suggests that local circulation along the mountain slopes may be moving anthropogenically contaminated air from lower altitudes to the higher altitude of the station site.

Finally, review of 1477 ozonesonde observations between December 1962 and December 1965 showed that 2 percent of the ozone concentration values exceeded 150 $\mu g/m^3$ (0.075 ppm).[112] However, of the 31 cases with such elevated concentrations, only three qualified as being unaffected by tropospheric source interferences. Thus only about 0.2 percent of the sample exceeded the 150-$\mu g/m^3$ (0.075-ppm) level.

The tropospheric ozone data appear to agree with the theoretical deductions both on the intensity of the stratospheric ozone impact and on the seasonal variation of such impact. Both bases of evidence suffer from uncertainties, and additional research studies are needed for a fuller and more confident assessment of such impact. Specific research needs include:

1. More definitive information on the frequency of occurrence and intensity of cyclogenetic events resulting in stratospheric ozone intrusion.
2. More definitive information on the ozone decay (and formation, if any) attending the subsidence of stratospheric ozone within the lower troposphere, especially within the planetary boundary layer.
3. Development of techniques for forecasting stratospheric ozone intrusions.
4. Further analysis of tropospheric ozone measurements to delineate the stratospheric and anthropogenic contributions to such ozone. The need here includes also the measurement of trace contaminants characteristic of anthropogenic sources.

The information and analysis presented in the preceding pages, although they do not answer all of the questions on the contribution of stratospheric ozone to ground level ozone concentrations, do provide important clarification. For example, it is now clear that violations of the oxidant/ozone NAAQS observed outside the smog season, particularly during the spring months, should not be attributed exclusively to anthropogenic causes. However, it is equally important that significant stratospheric ozone concentrations at ground level, though they may occur at any time, are less frequent during the summer months (that is, during the smog season). Within the summer months, such ozone is expected to occur at a background level somewhat lower than that of the annual average. Since the annual average ozone concentration has been estimated to be about 20 $\mu g/m^3$ (0.01 ppm) by Reiter[113] and about 60 $\mu g/m^3$ (0.03 ppm) by Mohnen[90] and Singh et al.,[126] it follows that the summer average could be 14 to 40 $\mu g/m^3$ (0.007 to 0.020 ppm) because of the spring-summer gradient. Data presented by Singh et al.[126] indicate that the 1-hr ozone concentrations in any given season in a remote atmosphere are typically about twice the ozone mean values. Thus an hourly ozone concentration range of 30 to 80 $\mu g/m^3$ (0.15 to 0.04 ppm) resulting from the stratospheric source alone can be expected in the summer season. The lower limit of 30 $\mu g/m^3$ (0.015 ppm) would thus be suggested by Reiter's analysis,[111] whereas Mohnen[90] and Singh et al.[126] would support the upper limit of 80 $\mu g/m^3$ (0.040 ppm).

Photochemistry of Natural Organics/NO_x

As in the case of stratospheric ozone, the role and importance of naturally emitted organics and NO_x in the formation of oxidant/ozone has been an issue.[43] The issue was originally raised as a result of early reports that the rates of organic emissions from vegetation are considerably higher, on a global basis, than those from manmade sources.[107]

Although natural emissions and anthropogenic emissions are, for the most part, geographically segregated, it is nevertheless reasonable to suspect that the much more abundant natural organics, emitted either within an urban or nonurban area, or brought in through transport, could contribute to ambient oxidant/ozone concentrations to an important degree. These suspicions became considerably stronger as a result of two recent findings: (a) The occurrence of a pervasive rural oxidant/ozone problem,[118] and (b) the high reactivity of the terpenes.[48,137] In either case, the finding could be interpreted to mean that natural organics may constitute a significant source of oxidant/ozone. As in the case of stratospheric ozone, the question regarding the role and importance of natural emissions as an oxidant/ozone source needs to be answered.

From a cursory examination of the current evidence, it becomes immediately apparent that certain questions regarding natural emissions have obvious answers or have been resolved by scientific evidence, whereas other questions appear unresolved. For example, it is certain that vegetation emits organic vapors and that some of these vapors (terpenes) play the dual role of ozone precursor and ozone scavenger. Questions that do not have obvious answers are: (1) What is the net effect on ozone concentrations of the atmospheric reactions of terpenes and of other natural organics? (2) What reactive organics, other than terpenes, are emitted by natural sources and at what rates?

Evidence pertinent to these questions is available and is fairly complete and definitive in some respects, but it is incomplete and ambiguous in others. The main source of ambiguity is that most researchers are, in general, relatively unfamiliar with natural organic emissions. Thus, unless convincing documentation is provided, results and conclusions on chemical identity, ambient concentrations, and emission and sink processes of natural organics are viewed with considerable skepticism. In the remainder of this section, a brief review is given of the relevant evidence available, and an attempt is made to interpret such evidence so as to provide the best possible judgment regarding the importance of natural emissions as an oxidant/ozone source.

There are two questions regarding the net impact of terpenes on ambient oxidant/ozone concentrations. First, what are the terpene concentrations actually observed in atmospheres with high oxidant/ozone levels? And second, what is the net effect of terpenes at such concentrations: Is it to produce or to destroy oxidant/ozone? The evidence available appears sufficiently complete and definitive[137] to resolve these questions.

Most of the qualitative analysis on organic emissions from vegetation has been done by Rasmussen and his coworkers. Chromatographic analysis by Rasmussen and Holdren[108] of ambient organics collected at remote sites showed the presence of 10 to 60 organic components in the C_5-C_{10} range; individual component concentrations were usually below 2 $\mu g/m^3$ (0.001 ppm). Hydrocarbon concentrations in rural areas have also been reported formally by Whitby et al.[141] and by Whitehead and Severs,[144] and informally by others.[89,117] However, individual organics were not identified in these studies. Because of this deficiency, and in view of the well documented occurrence of extensive urban pollutant transport, a natural origin for the organic compounds measured by these investigators cannot be supported. Nevertheless, their evidence leaves the question somewhat open. Some better documentation on natural organic compounds in ambient air was obtained by Westberg and Holdren.[137,140] Using a gas chromatograph linked to a mass spectrometer, these investigators identified in a forested area in Idaho, α-pinene, β-pinene, Δ-carene, and limonene present at concentrations from a few ppt for limonene to 730 ppt for β-pinene. Westberg and Holdren also measured rates of emission of these terpenes from the forest and found such rates to be consistent with their measurements of the ambient concentrations. The most significant finding from this study, however, was that air samples taken outside the forest canopy showed no measurable terpenes, suggesting an extremely short life for those organic compounds. Measurements by Lonneman et al.[82,83] in a forested area near Durham, North Carolina, showed the presence of a α-pinene, β-pinene, myrcene, and Δ-carene at concentrations from 0.3 ppb C (myrcene) to 90 ppb C (β-pinene)—roughly comparable to those reported by Westberg and Holdren. Other measurements in this same area showed the terpene concentrations to be at lower levels when the ambient ozone concentration was at higher levels. Finally, in addition to the monoterpenes already mentioned, isoprene was also found to be emitted by vegetation and to occur in ambient air.[104]

In conclusion, there is sufficient evidence to show that monoterpenes do occur in the ambient air but only within forested areas; in such areas, total terpene concentrations average 10 to 50 ppb C.[137] The evidence further shows that terpenes have extremely short lifetimes, such that, with the exception of isoprene, they are not transported downwind from their sources in appreciable quantities. Within urban areas, terpenes have not been detected. In their numerous efforts to search for and identify terpenes in urban atmospheres, EPA investigators only occasionally found isoprene, at ppb C concentrations. Thus the evidence does not support an important direct contribution of terpenes to urban oxidant/ozone concentrations; terpenes may contribute to the urban problem indirectly, however, through terpene-induced oxidant formation in rural areas. This possibility will be examined next.

Evidence of the potential of terpenes to form oxidant/ozone in rural areas has been observed both in laboratory and field studies. Smog chamber testing of terpene-NO_x mixtures showed clearly that terpenes are potent oxidant precursors and, more important, that relative to the less reactive hydrocarbons, their oxidant-forming potential is at a maximum at low hydrocarbon-to-NO_x ratios, typically at 2:1 to 2.5:1 molar concentrations.[48,140] The latter finding is significant because it means that in rural atmospheres with hydrocarbon-to-NO_x ratios typically much higher than 2.5:1, the terpenes could be effective as oxidant producers only partially, if at all. In fact, in the absence of sufficient NO_x, terpenes would act as ozone scavengers.[52] Under optimum hydrocarbon-to-NO_x ratio conditions, terpenes produced in smog chambers a maximum of 470 $\mu g/m^3$ (0.24 ppm) of oxidant/ozone for 5 ppm C of reactant terpene. Assuming a proportionality dependence of oxidant on organic reactant, it follows that at their typical ambient concentrations of 10 to 50 ppb C in the rural air, terpenes can produce no more than 2 to 4 $\mu g/m^3$ (0.001 to 0.002 ppm) of oxidant/ozone.[137,140]

The potential of rural-atmosphere organic compounds to produce oxidant was also explored in studies in which rural air samples were captured in plastic bags, spiked with various amounts of NO_x, and exposed to sunlight. Results by one investigator were interpreted to mean that in some rural areas, the ambient organic compounds have the potential to produce 40 to 120 $\mu g/m^3$ (0.02 to 0.06 ppm) of oxidant/ozone.[105] A recent study in rural Wisconsin showed that irradiation of captured air samples spiked with NO did not produce significantly more oxidant/ozone than the unspiked samples.[2] Finally, Singh et al.[126] analyzed ambient ozone monitoring data from a station at White River, Utah, and concluded that elevated levels of ozone in the summer occurred only when ambient NO_x was present at concentrations sufficiently high to act as photochemical ozone precursors (Figure 4-14). This evidence is suggestive but far from being conclusive insofar as the role and importance of natural organic compounds are concerned. First, it is extremely difficult to establish with confidence that oxidant/ozone formation in the captured rural air samples results solely from natural organic compounds; the presence of anthropogenic pollutants and of experimental artifacts cannot be ruled out.[137] Also, relative to free ambient air, captured air samples probably yield higher oxidant/ozone concentrations. Singh et al.[126] also agree that anthropogenic sources cannot be ruled out in explaining the elevated ozone concentrations observed in White River, Utah, in the summer (Figure 4-14).[126] Finally, substantial oxidant/ozone formation from natural organic compounds is not consistent with the laboratory evidence on terpenes, unless, of course, there are other yet unknown natural organic compounds with photochemical reactivities different from that of the terpenes. This latter possibility remains largely unexplored except for the case of methane, which is somewhat uncertain.

The modeling of the photochemical methane oxidation cycle in the lower troposphere has been a subject of considerable interest and controversy in recent years.[21,29,35,36,81] The importance of understanding the methane photo-oxidation cycle is not only relevant in its potential role as a source for natural background ozone concentration, but also in elucidating the half-lives and budgets for chemical species present in the atmosphere. Much of the debate has centered on whether or not the methane photo-oxidation cycle in the unpolluted troposphere represents a net ozone production or destruction process. This in turn reflects on the classical concept of ozone intrusion from the stratosphere as being the dominant source of surface ozone in the unpolluted troposphere.[90] Current models of the uncontaminated troposphere[2,81] are sufficiently sensitive to uncertainties in initial and boundary conditions, rate constants, transport parameters, and heterogeneous removal rates that quantitative statements regarding net ozone production or destruction are still tenuous. In the final analysis, the importance of modeling the clean troposphere

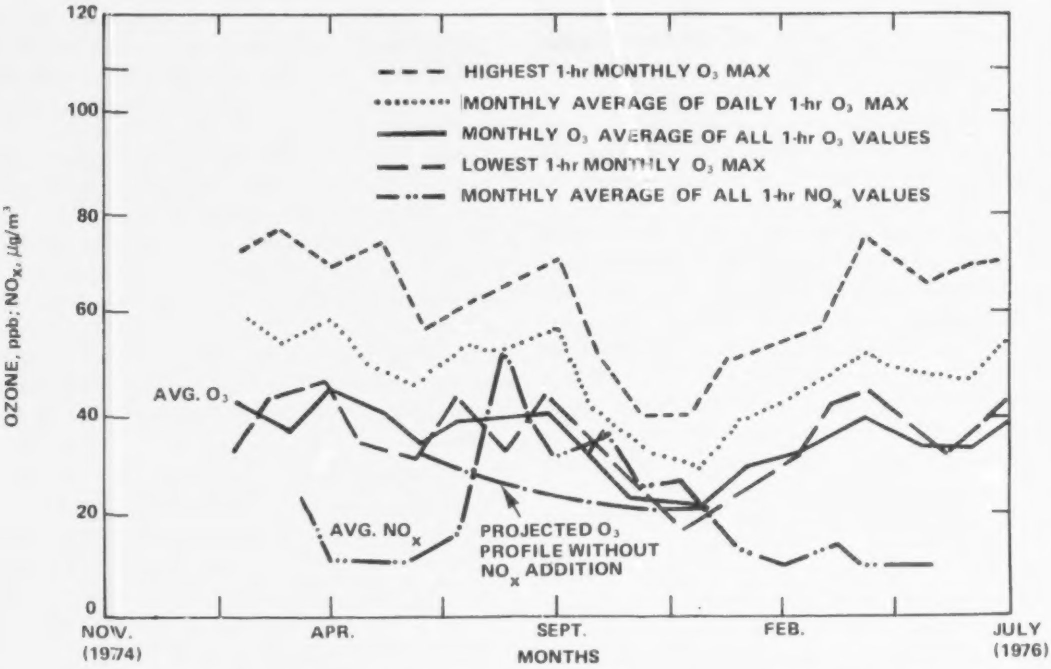

Figure 4-14. Effect of NO_x concentration increase on ozone formation at White River, Utah.[126]

resides in a clearer understanding of the physical and chemical processes in operation. This in turn will provide a better understanding of the effects of low-level anthropogenic contributions to rural oxidant/ozone contributions.

One may conclude that natural organic emissions (at least terpenes) do not directly affect oxidant/ozone formation within urban areas. Their indirect effects, however, are not as well understood. Natural organic compounds do occur in rural atmospheres and could possibly contribute to the large-area oxidant/ozone concentrations associated with high pressure episodes. Furthermore, products from the atmospheric degradation of terpenes could also contribute to those concentrations. The evidence available, however, suggests that within rural areas where ambient HC-to-NO_x ratios are high, terpenes act as scavengers rather than producers of ozone. Furthermore, terpenes decay too fast to survive transport into urbanized areas where the HC-to-NO_x ratios are more conducive to oxidant formation from terpenes. The limited evidence available suggests that chemical degradation products of terpenes are mainly particulates. Nevertheless, small amounts of gaseous products, including aldehydes, ketones, and organic acids are formed and could conceivably be transported into urban areas.[48] Considering all the evidence, there is no convincing reason to believe that natural organic emissions have an important impact on the oxidant/ozone-related air quality. Such a view is supported by some investigators[90] but not by others.[29]

SINKS OF OXIDANTS AND OF OXIDANT PRECURSORS

Introduction

The preceding sections identified and described the various sources of ambient oxidant/ozone and discussed their absolute and relative contributions to surface accumulation of oxidant/ozone. Implicit in those discussions was the assumption that observed concentrations of oxidant/ozone do not reflect source emission levels alone; rather, they reflect the composite impact of the source and sink processes. Not all sink processes have the same significance and role. For example, removal of ambient NO_2 in reactions forming PAN, nitrates, etc., is a sink process with entirely different significance than the removal of NO_2 on surfaces. Sink processes relevant to oxidant/ozone are those that directly or indirectly lessen the impact of the oxidant/ozone sources, thus reducing concentrations of ambient oxidant/ozone. Obviously, such sink processes include not only those for oxidant/ozone, but also those for oxidant/ozone precursors. For this reason, sink processes for hydrocarbons and NO_x will be discussed here rather than in the subsequent chapter on oxidant precursors.

Reviews of the various mechanisms by which pollutants are removed from ambient air have been made by Robinson and Robbins,[121] by Hidy,[63] and by Rasmussen et al.[109,110] The important mechanisms appear to be: (1) precipitation scavenging, including rainout (i.e., absorption in the cloud) and washout (i.e., capture by falling raindrops), (2) chemical reactions resulting in products that are not oxidants or that are rapidly removed from ambient air, (3) dry deposition (i.e., adsorption on aerosols and subsequent deposition on surfaces), and (4) adsorption on ground surfaces (soil, vegetation, etc.). Information on the occurrence of such processes for various pollutants exists, but it is mostly qualitative. For such information to be useful, it must be quantitative—that is, expressed in terms of data compatible with the available mathematical air quality simulation models. Such data, for example, are reaction rate constant and kinetic order values for the various chemical processes, and deposition rates for the various physical sink processes.

Sinks for Oxidants and Oxidant Precursors

Ozone is relatively insoluble in water. This, despite evidence that the oceans remove some ozone,[1] suggests that rainout and washout cannot be important sink processes for ozone.

Reaction of ozone with NO and with HC is an important sink process accounting for the complete removal of ozone from the surface air layers in urban areas at night. On-surface adsorption/destruction, and some destruction from locally emitted NO and HC account for the nighttime absence of ozone from the rural atmospheres at ground level. Thus, under ground-level conditions and at night, ozone appears to have a lifetime of only a few hours at most.

In air layers aloft, relatively free of fresh precursor and surface influences, ozone has been observed to remain extremely stable, at least during the night.[39] During the day, ozone is photolyzed rapidly by sunlight, but the resultant oxygen atoms react rapidly with molecular oxygen to regenerate the ozone. Thus sunlight has very little net effect on ambient ozone in the absence of other pollutants. In the presence of other pollutants at their global background levels, ozone participates in a photochemical destruction cycle resulting in an ozone half-life estimated to be 7 to 8 hr.[15] In the presence of other pollutants at or near urban concentrations, ozone is both produced and destroyed in such a way that the initial ozone loses its identity and significance. The ozone concentration that develops is the net result of the formation and destruction processes. The concentration and lifetime of such ozone depends on the composition and concentrations of the other reactants. From the evidence on long-range oxidant transport, it may be deduced that such transported ozone could have a lifetime of several days.

Chemical sink processes for ozone are qualitatively well established. Quantitatively, the processes occurring at ground level have not been defined from direct, real-atmosphere data because of the difficulty in isolating the chemical process from the on-surface physical processes. Nevertheless, reasonably accurate quantification has been achieved since the main ozone destruction/reaction steps are well defined kinetically and are included in the mathematical models relating air quality to emissions.

The dry deposition sink process for ozone is not well defined. Surfaces are known to act as ozone scavengers, but it is not certain that the composition and surface area of ambient aerosols are such that they cause substantial ozone destruction. The stability of ozone in polluted air layers aloft and in aerosol-containing smog chamber atmospheres suggests that removal of ambient ozone by aerosols cannot be an important sink process.

Absorption and/or destruction of ozone on ground surfaces (soil, vegetation, water, etc.) constitute a major sink for this gas on a global basis.[109] Measurements of uptake rates of different pollutants by certain forms of vegetation (e.g., alfalfa) showed the ozone rate to be comparable to that for NO_2 and somewhat lower than that for SO_2.[64] Recently, Garland and Penkett[47] reported ozone deposition velocities of 0.5 cm sec^{-1} for grass, 1.6 cm sec^{-1} for soil, and 0.4 cm sec^{-1} for sea water (compared to 2.8 cm sec^{-1} and 0.2 to 0.7 cm sec^{-1} for SO_2 for alfalfa and soils, respectively). These values are about three times lower than those reported by Hill,[64] but they are claimed to be more representative of average field conditions. At any rate, even the low values of

Garland and Penkett indicate a substantial removal of ozone by ground surfaces.

The evidence indicates that of the various possible sink processes for ozone, chemical destruction, chemical reaction, and on-surface destruction are probably the most important. The importance of the surface sinks, however, can be assessed only through use of models. One air quality simulation model has been used to compute ozone concentrations in a moving polluted air parcel with and without the incorporation of surface losses.[75] Results showed that with representative surface loss rates for ozone and NO_2 included, the air parcel at the end of an 8-hr trajectory was computed to have an average ozone concentration 25 percent lower than that with the surface losses excluded.

Information on sink processes for oxidants other than ozone is limited to PAN and NO_2. Judging from PAN solubility in water, rainout and washout are not expected to be significant sinks for PAN. The reaction of PAN with NO (0.16 ppm min^{-1}) is some two orders of magnitude slower than the reaction of ozone with NO; nevertheless, this sink process could be important in urban areas during evening hours when NO accumulates to high concentrations. PAN is not destroyed at a significant rate by sunlight photolysis ($k = 5 \times 10^{-3}$ hr^{-1})[4] but is subject to reversible thermal decomposition into NO_2[59] (see also section on atmospheric reaction mechanisms). Because of this latter process, any removal process for NO_2 will also constitute an indirect sink for PAN. The low vapor pressure of PAN suggests that dry deposition could be an important sink for this oxidant; however, quantitative data are not available. In contrast, data for on-surface adsorption do exist and show PAN to be somewhat less adsorbable than ozone, with a deposition velocity of about 0.25 cm sec^{-1} for grass and soil surfaces.[47] Such a value suggests that removal on surfaces is probably one of the major sinks for PAN in the lower troposphere. In air layers aloft, where the NO-reaction and surface sinks are ineffective, PAN can travel long distances.

Nitric oxide and nitrogen dioxide can be removed to varying degrees by all four sink mechanisms. The primary sink for NO is the reaction with oxidizing radicals to produce NO_2. Rainout and washout removal of NO are minimal because of its low solubility. Adsorption of NO by vegetation and by soil occurs very slowly, at deposition velocities around 0.1 cm sec^{-1}.[109] No_2 is removed chiefly by oxidation into nitrates and peroxyacylnitrates and subsequent removal through precipitation, dry deposition, and surface adsorption. Removal of NO_2 on alfalfa surfaces was measured and found by Hill[64] to occur with a deposition velocity of 2 cm sec^{-1}. The impact of surface sinks for NO_2 on ambient ozone and NO_2 was computed by Killus and Jerskey.[75]

Information on sinks for HC is far more incomplete than for the other oxidant-related pollutants, probably because of the large variety of organic compounds (hydrocarbon and non-hydrocarbon) in ambient air. Hydrocarbons are, in general, insoluble in water and therefore cannot be removed significantly by precipitation. Chemical reactions converting the reactive HC to soluble and/or condensable products constitute the most important sink processes. Limited data reported by Hidy[63] indicate that 1 to 10 percent (by weight) of reactive HC emissions are converted to removable aerosol. The remaining HC are eventually oxidized to CO_2 and H_2O, but with parallel formation of oxidant/ozone. Such a conversion does not, therefore, constitute a sink. Removal by soil apparently does occur, but data exist for a few hydrocarbons only (e.g., ethylene and acetylene).[109] The sink processes for unreactive hydrocarbons are the same, in general, as for the reactive ones, but they occur more slowly.

SUMMARY

Evidence shows that in and around urban centers with high oxidant/ozone concentrations, photochemical oxidant and ozone are mainly formed from anthropogenic organic and NO_x emissions. In the years since publication of the predecessor to this criteria document, the mechanisms of the atmospheric oxidant/ozone formation process have been studied intensively and are now better understood. Most noteworthy are recent findings pertaining to the roles of hydroxyl ($OH\cdot$) and ($HO\cdot_2$). The reaction with OH has been established to be a major HC-consuming process, and OH and HO_2 have been identified as having major roles in the atmospheric oxidation of NO to NO_2. Aldehydes and PAN were also found to play important roles in the atmospheric reaction. For olefins and paraffins, at least, these reactions are now understood to the extent that the kinetics of photochemical hydrocarbon-NO_x reaction systems, as observed in the laboratory, can be described with reasonable accuracy. Additional research is needed to understand the atmospheric reactions of aromatic hydrocarbons and to further clarify the differences between laboratory and ambient atmospheric chemical systems.

The photochemical formation of oxidant/ozone is the result of two coupled processes: a physical process involving dispersion and transport of the oxidant precursor emissions (e.g., HC and NO_x) and the photochemical reaction process. Both processes are strongly influenced by meteorological factors such as dispersion, solar radiation, temperature, and humidity. Recently compiled statistics on wind velocity and mixing height showed that episodes of limited dispersion are most common in the Far West and within the Rocky Mountain region, least common over the Plains States, and of intermediate frequency east of the Mississippi. New measurements of solar radiation lead to results somewhat different from those reported earlier. Recent field and laboratory studies suggest that at temperatures below approximately 55° to 60°F (13° to 16°C), concentrations of photochemical ozone are unlikely to exceed the national 1-hr 160-$\mu g/m^3$ (0.08-ppm) standard.

Of all recent developments regarding the understanding of the sources of photochemical oxidant/ozone, perhaps the most important ones were in the area of photochemical oxidant/ozone transport. Short-range (urban-scale) transport has been shown to occur and to cause the highest ozone concentrations some distance downwind from the core area (region of highest emission) of urban center. Intermediate-range (mesoscale) transport has been identified in the form of urban oxidant/ozone plumes extending as far as 100 miles (160 km) or more downwind, and also involved in land-sea-breeze circulation. Finally, synoptic-scale transport (over a range of several hundred miles) associated with high pressure systems has been found to occur extensively. These findings have significant implications with respect to the location of oxidant/ozone monitors.

Conditions under long-range transport are such that ozone production per unit of precursor is enhanced. Also, many organics previously thought to be unreactive are now believed to have significant ozone-producing potential.

In addition to the tropospheric photochemistry of anthropogenic emissions, potential sources of tropospheric oxidant/ozone are the stratosphere and the photochemistry of natural organic and NO_x emissions. Estimates of stratospheric ozone intrusions at ground level are based on two types of evidence: (1) global circulation patterns, namely, patterns in air interchange between stratosphere and tropsphere, and (2) data on variations of ozone concentrations in remote rural areas. Based on the evidence of stratosphere-troposphere interchange, the annual average stratospheric contribution to ozone concentrations at ground level is estimated to be 0.022 to 0.05 ppm. The highest concentrations, at or above 0.08 ppm, from that source are expected to occur mostly during April and May. Occurrence of such major intrusions of stratospheric ozone concentration during the spring months in midlatitudes was indicated also by analysis of the ozone data for remote rural areas. The evidence suggests that the probable concentration of stratospheric ozone reaching ground level during the smog season (usually late summer or early fall) is about 60 $\mu g/m^3$ (0.03 ppm). There are also more recent data obtained at Whiteface Mountain, New York, suggesting a maximum 24-hr concentration of 72 $\mu g/m^3$ (0.037 ppm) stratospheric ozone in July.

Certain organic emissions from vegetation (terpenes) were found to play the dual role of ozone precursor and destroyer. Despite the substantial rates at which they are emitted in forested areas, the ambient concentrations of such organics, because of their reactivity and the areal dispersion of their sources, seldom exceed a few parts per billion. At these ambient concentrations, the *direct* potential of terpenes for photochemical ozone formation is estimated to be negligible. It is conceivable, however, that the products of atmospheric reactions involving large amounts of terpenes do have a significant impact on oxidant/ozone-related air quality.

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5. OXIDANT PRECURSORS

INTRODUCTION

As has been established, most of the oxidants found in urban atmospheres are secondary pollutants, and the primary pollutants that act as oxidant precursors are the hydrocarbons and the nitrogen oxides (i.e., nitric oxide and nitrogen dioxide, NO and NO_2). Not all atmospheric hydrocarbons are significant oxidant producers. Methane is the most important exception. In addition, there are nonhydrocarbon organics (e.g., aldehydes) that are also capable of forming oxidants. However, the term "hydrocarbons," or, specifically, "nonmethane hydrocarbons," best describes the organic component of the oxidant precursor mixture in the atmosphere.

Data on the nature, sources, concentration, and the concentration variation patterns for the oxidant precursors are discussed in this chapter. Emphasis is placed on the organic precursors, as the NO_x data are presented in detail in the literature.[22] The quantitative relationships between oxidant and oxidant precursors are treated in a separate chapter.

AMBIENT LEVELS AND VARIATIONS OF OXIDANT/OZONE PRECURSORS

Organic Compounds in Urban Atmospheres

Information on the nature and relative amounts of the various organic compounds present in polluted atmospheres is needed to identify pollutants that have direct, adverse effects on human health and/or welfare. It is also needed to provide direct estimates, however rough they may be, of the levels of controllable and noncontrollable emissions. Data on the composition of polluted atmospheres are also necessary for characterizing in terms of their oxidant-producing potential the emissions discharged from various sources. A large part of the information needed can be obtained through analysis of the ambient air. The composition of emissions can also be determined from measurements at the sources. It is only from the combined use of such ambient air and source emission data that the impact of organic emissions on air quality can reliably and comprehensively be assessed.

Most of the detailed studies of organic compounds in the urban atmosphere were conducted during the 1960's. The results were summarized and discussed in the criteria document for hydrocarbons.[60] Those studies and other more recent ones identified a large number of hydrocarbons (Table 5-1)[4,62,78] and some aldehydes in the urban atmosphere.[8] It is almost certain that such identification is far from complete because of the analytical problems created by the enormous complexity of the ambient organic mixtures and the trace concentrations of the various components. The composition of organics in urban atmospheres differs from city to city and reflects emission composition and the influences of chemical reaction. For example, industrial emissions and chemical reactions cause higher relative levels of paraffins and of other nonreactive organics in the ambient air.[45,51] Automobile emissions usually dominate the composition of organics in urban ambient atmospheres, especially during the peak traffic hours.[45] Some stationary emission sources, however, can be important also. More detailed but indirect information on the composition of organics likely to be present in urban ambient atmospheres has been provided by analysis of source emissions. Analyses of the exhaust and evaporative emissions from gasoline-fueled automobiles have identified some 200 hydrocarbons[30] and several oxygenated hydrocarbons,[76] many of which could not be detected in the ambient air, although they were certain to be present. Data are also available in varying degrees of detail and specificity on the composition of organic emissions from diesel-powered automobiles (Figure 5-1),[39] from aircraft (Figure 5-2),[21] from natural gas lines, and from solvent evaporation. The presence of these latter organic emissions in the ambient air and, in fact, their quantitative contributions to the ambient organic load have been explored by investigators[53,79] with some definitive results. Contributions, for example, have been estimated by Mayrsohn and Crabtree in their source study (reconciling atmospheric hydrocarbon data with known hydrocarbon sources and their emissions) of the Los Angeles atmosphere.[53] Mayrsohn's conclusions were that (1) automotive exhaust accounts for somewhat less than 50 percent of the nonmethane hydrocarbons; (2) gasoline and gasoline vapor together constitute the second largest source of atmospheric hydrocarbons, totaling 30 to 35 percent by weight; (3) commercial and geogenic natural gas makes up about 20 percent of the total by weight; and (4) other emissions, such as those from solvent evaporation, diesel and aircraft emissions, and refrigerant and propellent usage are also present so that the percentage contributions attributed to the preceding three sources should be regarded as upper limits.

Probably not all organics emitted by the various sources will be found intact in the ambient air. Many may be removed through sink processes (e.g., deposition on aerosol and ground surfaces and dissolution in water) before they can participate substantially in atmospheric reaction processes. Considering this latter uncertainty and the limited compositional data available for most source emissions (e.g., for the exhausts from gasoline- and diesel-powered automobiles and from aircraft), one can only assume that current knowledge of the composition of the organics in the urban atmosphere is incomplete and, therefore, that the search for and identification of unknown organic air pollutants should continue.

TABLE 5-1. HYDROCARBONS IDENTIFIED IN AMBIENT AIR[4,62,78]

Carbon number	Compound
1	Methane
2	Ethane
	Ethylene
	Acetylene
3	Propane
	Propylene
	Propadiene
	Methylacetylene
4	Butane
	Isobutane
	1-Butene
	cis-2-Butene
	trans-2-Butene
	Isobutene
	1,3-Butadiene
5	Pentane
	Isopentane
	1-Pentene
	cis-2-Pentene
	trans-2-Pentene
	2-methyl-1-Butene
	2-methyl-2-Butene
	3-methyl-1-Butene
	2-methyl-1,3-Butadiene
	Cyclopentane
	Cyclopentene
6	Hexane
	2-Methylpentane
6 (cont.)	3-Methylpentane
	2,2-Dimethylbutane
	2,3-Dimethylbutane
	cis-2-Hexane
	trans-2-Hexane
	cis-3-Hexane
	trans-3-Hexane
	2-methyl-1-Pentene
	4-methyl-1-Pentene
	4-methyl-2-Pentene
	Benzene
	Cyclohexane
	Methylcyclopentane
7	2-Methylhexane
	3-Methylhexane
	2,3-Dimethylpentane
	2,4-Dimethylpentane
	Toluene
8	2,2,4-Trimethylpentane
	o-Xylene
	m-Xylene
	p-Xylene
9	*m*-Ethyltoluene
	p-Ethyltoluene
	1,2,4-Trimethylbenzene
	1,3,5-Trimethylbenzene
10	*sec*-Butylbenzene

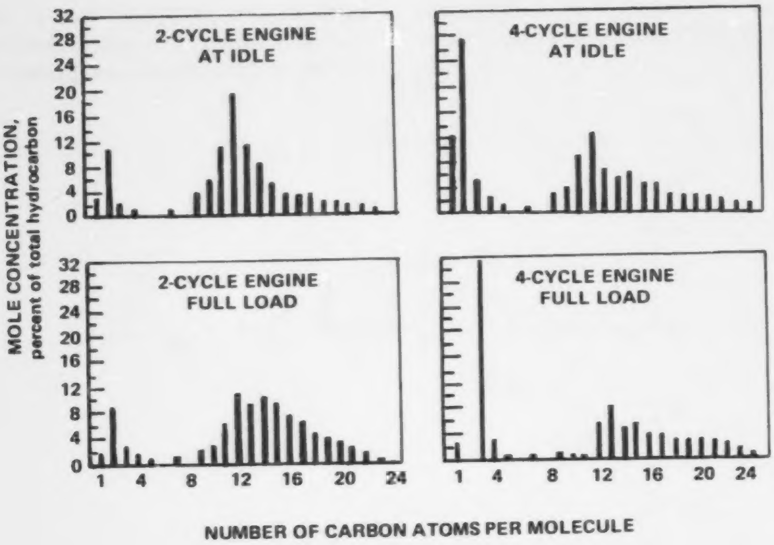

Figure 5-1. Distribution of hydrocarbons in diesel exhaust gas.[39]

Data on total levels of organic compounds in urban atmospheres are relatively abundant, but most of these data suffer from considerable measurement error (see Chapter 7). Some statistics on such data are shown for six urban centers in Table 5-2.[24] Total organic concentrations in ambient air are usually reported as totals of nonmethane hydrocarbons and as averages over the 6- to 9-a.m. period. Methane is excluded because it is nonreactive and chiefly of natural origin, and because its concentration usually overwhelms that of all other organics combined. The 6- to 9-a.m. period has been chosen and adopted in the early efforts by the Federal government to develop a model relating air quality to emissions (observational model).[22] The rationale behind that choice was that ambient concentrations during the early morning hours are a measure of the strength of the emissions only, as they are virtually unaffected by the chemical sinks. Specifically, the 6- to 9-a.m. concentrations reflect mainly the vehicular emissions, and such concentrations are relatively unaffected by changes in emissions from stationary sources. Some recent evidence suggests that a given amount of precursors, when injected into the reaction system, gradually, throughout the morning and afternoon, produce essentially the same ozone concentration as the same amount of precursors injected during the 6- to 9-a.m. period.[42] Such evidence was obtained from smog chamber studies and should be verified.

The concentration levels listed in Table 5-2 are typical of those in ordinary urban atmospheres, but they can be considerably higher. In Los Angeles, for example, total hydrocarbon concentrations have been as high as 30 ppm. This level is equivalent to a nonmethane hydrocarbon total of greater than 10 ppm.[16] In addition to the nonmethane organics upon which local contributions are superimposed methane also is present at a global background concentration of about 1.4 ppm.[10]

Seasonal and diurnal patterns of variation in nonmethane total organic levels have been discussed in the literature.[23,60] Briefly, seasonal variations have not been well established for lack of adequate data. In 14 of the 17 California cities for which adequate data exist, the highest hydrocarbon concentrations occurred in October or November. Such consistency is presumably a consequence of the generally similar meteorological conditions from year to year along the California coast. Cities elsewhere would be expected to show other patterns, depending on their particular meteorology. Diurnal patterns observed in many cities show two distinct daily peaks, one during 6- to 9-a.m., and another broader peak in the afternoon hours. Both peaks presumably reflect the automobile traffic and local meteorological dispersion patterns.

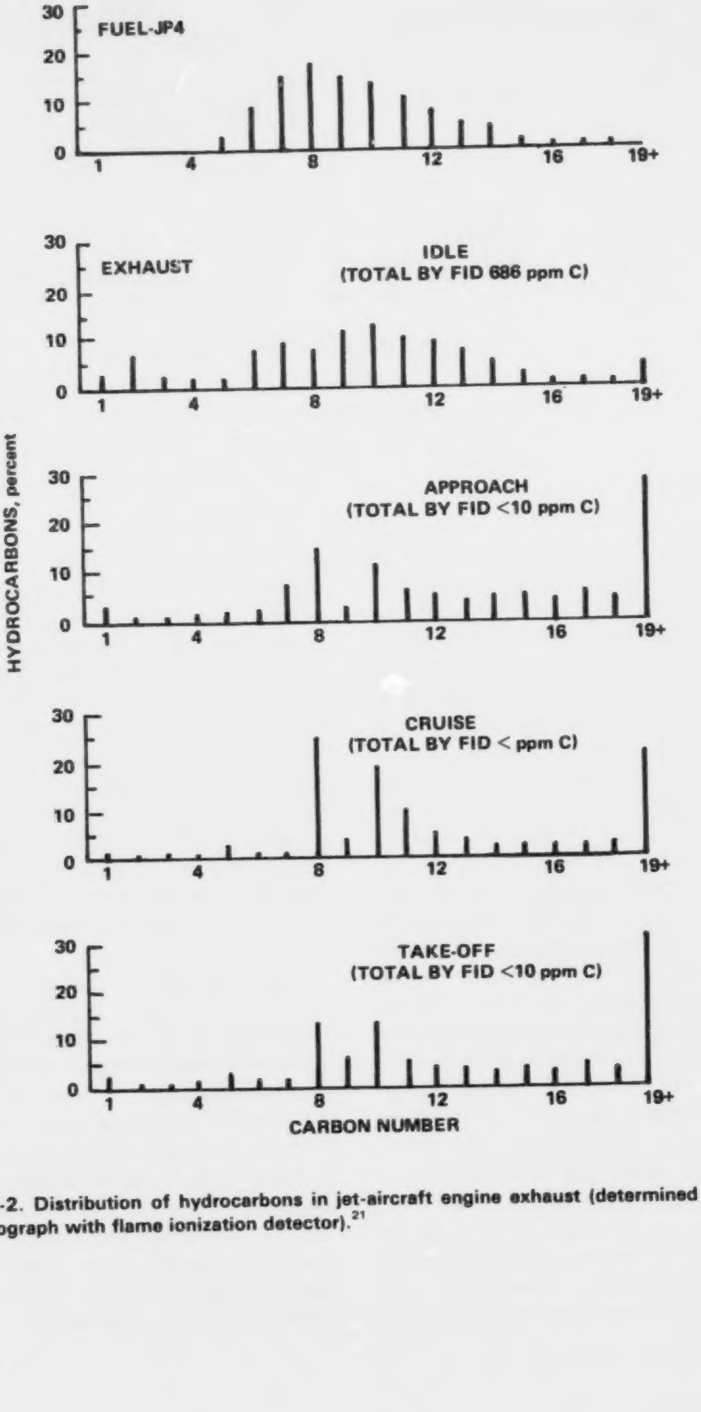

Figure 5-2. Distribution of hydrocarbons in jet-aircraft engine exhaust (determined by gas chromatograph with flame ionization detector).[21]

TABLE 5-2. FREQUENCY DISTRIBUTIONS FOR 6-TO 9-a.m. NONMETHANE HYDROCARBON CONCENTRATIONS AT CAMP SITES, 1967-72[24]

(ppm C)

				Percentile[a]										Arithmetic		Geometric	
Site	Year	Number of samples	Min.	10	20	30	40	50	60	70	80	90	Max.	Mean	Std. dev.	Mean	Std. dev.
Denver, Colo. (060680002A10)	67[b]	29	0.0	0.0	0.3	0.4	0.6	0.9	1.1	1.8	2.3	2.7	5.2	1.3	1.2	0.74	3.69
	68[b]	161	0.0	0.2	0.4	0.6	0.8	1.0	1.1	1.4	1.7	2.7	5.3	1.2	1.0	0.87	3.62
	69[b]	219	0.0	0.0	0.1	0.3	0.5	0.7	0.8	1.0	1.3	1.7	4.9	0.8	0.8	0.50	3.26
	70[b]	231	0.0	0.0	0.2	0.3	0.5	0.6	0.8	1.0	1.1	1.6	5.9	0.8	0.7	0.52	2.96
	71[b]	178	0.0	0.3	0.6	0.7	0.9	1.1	1.3	1.5	1.8	2.4	5.7	1.3	0.9	0.99	2.29
	72	282	0.0	0.1	0.4	0.5	0.7	0.9	1.1	1.3	1.6	2.0	3.9	1.0	0.7	0.74	2.73
Washington, D.C. (090020002A10)	66[b]	250	0.0	0.0	0.1	0.2	0.3	0.4	0.5	0.6	0.7	1.0	1.8	0.5	0.3	0.35	2.56
	68[b]	244	0.0	0.0	0.1	0.1	0.3	0.4	0.5	0.6	0.8	1.1	2.6	0.5	0.4	0.35	2.73
	69[b]	231	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.3	0.3	0.5	2.3	0.2	0.2	0.16	2.27
	70[b]	267	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.3	0.5	0.8	2.0	0.3	0.3	0.19	2.62
	71	279	0.0	0.0	0.0	0.0	0.1	0.1	0.2	0.3	0.5	0.8	3.3	0.3	0.3	0.20	2.64
	72[b]	187	0.0	0.0	0.0	0.0	0.1	0.1	0.3	0.5	0.6	1.1	2.0	0.4	0.4	0.25	2.82
Chicago, Ill. (141220002A10)	68[b]	146	0.0	0.1	0.2	0.4	0.6	0.8	1.0	1.1	1.4	1.8	2.8	0.8	0.6	0.64	2.58
	69	300	0.0	0.0	0.1	0.3	0.5	0.7	0.8	1.1	1.4	1.8	3.6	0.8	0.7	0.54	2.99
	70[b]	220	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.5	0.8	1.4	4.1	0.5	0.7	0.25	3.20
	71[b]	269	0.0	0.0	0.2	0.3	0.4	0.5	0.6	0.8	1.0	1.4	2.7	0.6	0.5	0.46	2.58
Philadelphia, Pa. (397140002A10)	66[b]	142	0.0	0.0	0.3	0.5	0.6	0.7	0.8	1.1	1.4	1.7	4.2	0.9	0.7	0.64	2.70
	67[b]	260	0.0	0.0	0.1	0.2	0.3	0.5	0.6	0.8	1.1	1.6	5.9	0.7	0.7	0.42	3.11
	68[b]	154	0.0	0.0	0.0	0.1	0.2	0.3	0.5	0.6	0.9	1.6	3.7	0.6	0.7	0.34	3.17
	69[b]	192	0.0	0.0	0.1	0.2	0.3	0.5	0.6	0.7	0.9	1.3	3.7	0.6	0.5	0.42	2.74
	70[b]	264	0.0	0.0	0.1	0.2	0.3	0.4	0.6	0.7	1.0	1.3	2.3	0.5	0.4	0.37	2.90
	71[b]	234	0.0	0.0	0.0	0.1	0.2	0.2	0.4	0.4	0.6	0.9	3.0	0.4	0.4	0.27	2.74
	72[b]	59	0.0	0.0	0.0	0.1	0.3	0.4	0.4	0.6	0.7	1.1	2.4	0.5	0.4	0.31	3.02
Cincinnati, Ohio	68[b]	109	0.0	0.0	0.0	0.1	0.2	0.3	0.4	0.6	0.8	1.4	3.4	0.5	0.6	0.31	3.18
	69[b]	188	0.0	0.0	0.0	0.2	0.2	0.4	0.4	0.5	0.7	1.1	3.1	0.5	0.5	0.32	2.79
	70[b]	99	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.4	2.3	0.1	0.3	0.23	2.22
	71[b]	104	0.0	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.9	1.1	2.0	0.6	0.4	0.45	2.37
	72[b]	238	0.0	0.1	0.2	0.3	0.3	0.4	0.4	0.5	0.7	1.0	3.4	0.5	0.4	0.40	2.09
St. Louis, Mo. (264280002A10)	68[b]	198	0.0	0.0	0.0	0.2	0.4	0.6	0.8	0.9	1.1	1.6	4.4	0.7	0.7	0.44	3.25
	69	292	0.0	0.0	0.1	0.2	0.3	0.4	0.5	0.7	1.0	1.4	3.4	0.5	0.5	0.37	2.83
	70[b]	232	0.0	0.0	0.0	0.1	0.2	0.3	0.4	0.6	0.7	1.1	2.9	0.4	0.4	0.31	2.72
	71	314	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.3	0.4	0.7	2.5	0.2	0.3	0.19	2.54
	72[b]	235	0.0	0.0	0.0	0.0	0.1	0.2	0.2	0.3	0.4	0.7	1.8	0.3	0.2	0.21	2.42

[a] Concentrations greater than or equal to specified value in indicated percentage of samples.

[b] Yearly standard exceeded.

Since hydrocarbon emission controls went into effect in the 1960's, ambient air hydrocarbon concentrations have been observed to trend downward with time in some urban areas[82] but not in others.[80] Figure 5-3 shows such a downward trend for the Los Angeles atmosphere.[40] The trend is probably a real one, even though the nonmethane hydrocarbon data used to construct these trend plots were obtained indirectly (that is, they were computed from total hydrocarbon data and data on established relationships between total and nonmethane hydrocarbon for the Los Angeles atmosphere). Such data, as already stated, reflect control of automotive emissions only. In areas where substantial controls have been applied to stationary sources also, the impact of these latter controls on daily maximum or 6- to 9-a.m. hydrocarbon concentrations will not be detectable; however, a detectable impact on the 24-hr average hydrocarbon concentrations may be observed. In general, the analysis and interpretation of ambient air hydrocarbon data to assess the impact of emission controls should be done with great care to avoid biases and, hence, misleading results. Such biases can be introduced, for example, by ambient air measurement errors, by inappropriate definition of the ambient concentration, and by the disproportionate influence of a single emission source on measurements made at an improperly located measurement station.

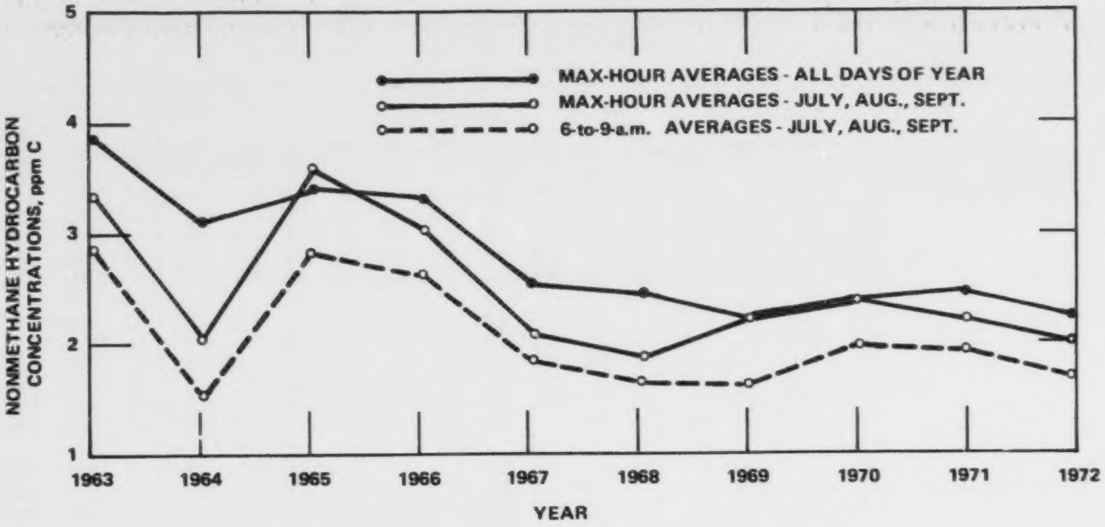

Figure 5-3. Nonmethane hydrocarbon trends in Los Angeles, 1963-1972.[62]

Organic Compounds in Rural and Remote Areas

Occurrences of ambient organic compounds in rural and remote areas are of interest only to the extent that they constitute evidence on the role of natural organics in the ambient air oxidant problem.[28] Since such evidence has already been discussed (in the chapter on the natural sources of oxidant/ozone) and the conclusion was reached that the terpenes and methane have no important role, the information to be presented in this section will serve merely to provide additional detail and documentation for that previous discussion and conclusion. Specifically, more complete and detailed evidence will be presented here on the occurrence of naturally emitted organics in rural and remote areas, but with the clear understanding that such evidence relates to occurrence alone and not to the impact of such organics on oxidant air quality.

Besides the ubiquitous anthropogenic pollutants, rural atmospheres also should contain natural organics such as methane and organics emitted by vegetation. Several of these latter organics have been identified either directly, through ambient air analysis, or indirectly, through analysis of the material emitted by vegetation samples processed in the laboratory.[68] Organic compounds found by Rasmussen to be emitted by vegetation are listed in Table 5-3.[68] This list is by no means complete, since not all types of vegetation were examined. Studies reported in the Russian literature indicate that a considerable number of plant species release low molecular weight hydrocarbons and aldehydes and a wide variety of essential oil components.[41]

TABLE 5-3. VOLATILE PLANT PRODUCTS IDENTIFIED BY RASMUSSEN[68]

Natural organic compounds		
α-Pinene	Santene	α-Ionone
β-Pinene	Camphene	β-Ionone
Myrcene	*n*-Heptane	α-Irone
D-Limonene	Isoprene	

Aside from the organics clearly associated with emissions from vegetation, measurements in remote areas have also shown the presence of organics that are commonly, but apparently not exclusively, associated with anthropogenic activities. For example, at Point Barrow, Alaska, Cavanagh et al. found benzene, pentane, butane, ethane, ethylene, acetaldehyde, and acetone at sub-ppb levels (acetone at about 100 ppb) and attributed their origin to biological processes.[19]

A few data on total organic concentrations in rural and remote atmospheres are available, but here again, as in the case of the urban atmospheres, most of the existing data are unreliable. One cause of unreliability is the relatively large error associated with the measurement of total non-methane organics. An additional problem, however, with the rural and remote atmospheric data arises from the lack of evidence on the composition of the organic pollutant mixture and, specifically, on the relative levels of vegetation-related and anthropogenic pollutants. This lack in compositional information has two consequences. First, it does not permit proper calibration of the ionization detector of the hydrocarbon analyzer, resulting in additional measurement error. Second, it attributes erroneously high concentration to the natural organics, since corrections for the anthropogenic components are not possible. Probably it is partly because of such errors that some of the reported values for total natural organic concentrations have been as high as 5 to 10 ppm C.[85] Extensive measurements in rural areas by EPA and other investigators using sampling and analysis procedures carefully designed to minimize such errors did not show the total of natural organics to be more than 0.1 ppm C.[69,84] Whenever and wherever concentrations were higher, there was invariably evidence of anthropogenic contamination.[50,72] Finally, though it is probable that there are natural organic pollutants as yet unidentified, there are no strong indications that such unknowns could drastically change the current view regarding the role of natural organics in the oxidant/ozone problem. Such a role does not appear to be an important one, as was concluded also from the discussions in the preceding chapter dealing with the natural sources of ambient air oxidants.

Nitrogen Oxides

Information on the sources, concentrations, and variations in concentrations of nitrogen oxides has been reported elsewhere in detail.[2,26] The information presented here is limited to data on ambient concentrations in urban and rural/remote atmospheres and is intended merely to complement the information on oxidant and oxidant precursor concentrations.

Tables 5-4 and 5-5 include data on ambient NO and NO_2 concentrations in seven urban centers outside California.[24] Data from selected California sites, where concentrations levels are generally higher, are shown in Tables 5-6 and 5-7.[2] Such data show a slight upward trend with time, as illustrated in Figure 5-4.[16]

Concentrations of NO and NO_2 in rural and remote areas are extremely low, the average levels being at or below the limit of detectability for current monitoring instruments. Thus, early measurements of the NO_2 background concentration showed levels generally below 5 ppb.[43,49] More recent measurements of NO and NO_2 in rural areas in Ohio, Maryland, and Pennsylvania indicated NO and NO_2 levels around 5 ppb, with a few excursions to as high as 12 ppb.[71,72] Furthermore, it appears that the concentration of NO_x in an urban air mass decays rapidly as the air mass moves away from the city. This is illustrated by ambient NO_x data taken within and outside Dayton, Ohio. Such data showed the city core levels of NO_x to range from 75 ppb to 450 ppb, whereas the concentrations at a site 18 miles from the downtown area ranged only from 8 to 80 ppb.[77]

The extremely low levels of NO in the rural and remote areas or, alternatively, the lack of adequately sensitive analytical instruments, is a problem in that it limits the conclusiveness of the field studies on oxidant. Thus at present, it is not possible to establish reliably the levels of natural NO_x and hence to estimate the relative contributions of the natural and manmade sources to rural NO_x. This uncertainty, in view of the important role of the NO_x factor in rural oxidant formation (see Chapter 4) makes it difficult to delineate completely and clearly the relative roles of anthropogenic hydrocarbon and NO_x as precursors in the atmospheric oxidant/ozone formation process.

SOURCES OF OXIDANT PRECURSORS

Introduction

Hydrocarbons and nitrogen oxides are emitted into the atmosphere from both natural and manmade sources, with the contribution of natural sources being the greater. However, the larger contribution by the natural sources does not significantly influence the oxidant problem, since the natural and anthropogenic emissions are geographically segregated, with the anthropogenic ones concentrated in the populated areas. Natural hydrocarbon emissions arise mostly from biological processes, trees, and localized sources such as seepage from natural gas and oil fields. Of the anthropogenic sources, combustion of fuels is

TABLE 5-4. FREQUENCY DISTRIBUTION DATA FOR 6-TO 9-a.m. NITRIC OXIDE CONCENTRATIONS AT CAMP SITES, 1962-72[24]

(ppm)

Site	Year	Number of samples	Min.	Percentile[a]									Max.	Arithmetic		Geometric	
				10	20	30	40	50	60	70	80	90		Mean	Std. dev.	Mean	Std. dev.
San Francisco, Calif. (056860002A10)	62[b]	112	0.00	0.00	0.00	0.01	0.01	0.02	0.02	0.03	0.04	0.06	0.13	0.02	0.02	0.02	2.51
	63[b]	179	0.00	0.00	0.00	0.01	0.02	0.02	0.03	0.05	0.06	0.08	0.32	0.03	0.03	0.02	2.98
	64[b]	13	0.01	0.01	0.02	0.02	0.03	0.04	0.04	0.05	0.06	0.20	0.29	0.07	0.08	0.04	2.63
Denver, Colo. (060580002A10)	65[b]	223	0.00	0.01	0.02	0.03	0.03	0.04	0.05	0.06	0.08	0.11	0.31	0.05	0.04	0.04	2.14
	66	297	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.11	0.15	0.30	0.06	0.06	0.05	2.61
	67[b]	191	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.07	0.09	0.13	0.32	0.06	0.05	0.05	2.31
	68[b]	192	0.00	0.01	0.02	0.02	0.04	0.05	0.06	0.07	0.10	0.04	0.27	0.06	0.05	0.04	2.71
	69	285	0.00	0.01	0.02	0.03	0.04	0.05	0.07	0.09	0.12	0.16	0.45	0.07	0.07	0.05	2.76
	70[b]	252	0.00	0.02	0.03	0.04	0.06	0.07	0.09	0.11	0.13	0.16	0.42	0.08	0.06	0.06	2.52
	71[b]	184	0.00	0.02	0.03	0.04	0.05	0.07	0.08	0.10	0.12	0.16	0.40	0.08	0.07	0.06	2.38
	72[b]	241	0.01	0.02	0.04	0.05	0.07	0.08	0.10	0.12	0.15	0.22	0.44	0.10	0.08	0.08	2.32
Washington, D.C. (090020002A10)	62	289	0.00	0.01	0.01	0.02	0.02	0.03	0.04	0.05	0.06	0.10	0.32	0.04	0.04	0.03	2.53
	63	279	0.00	0.00	0.01	0.02	0.02	0.04	0.05	0.06	0.08	0.13	0.40	0.06	0.06	0.04	2.82
	64	310	0.00	0.00	0.01	0.01	0.02	0.03	0.04	0.05	0.08	0.14	0.66	0.05	0.07	0.03	3.11
	65	274	0.00	0.00	0.01	0.02	0.02	0.03	0.04	0.06	0.08	0.12	0.38	0.05	0.06	0.03	2.83
	66	310	0.00	0.01	0.01	0.02	0.03	0.04	0.04	0.05	0.07	0.12	0.53	0.05	0.06	0.04	2.66
	67	304	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.10	0.16	0.74	0.07	0.08	0.05	2.74
	68[b]	270	0.00	0.01	0.02	0.03	0.03	0.04	0.05	0.06	0.08	0.13	0.49	0.06	0.06	0.04	2.56
Washington, D.C. (090020003A10)	69[b]	269	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.07	0.08	0.14	0.62	0.07	0.08	0.05	2.73
	70	299	0.00	0.01	0.02	0.03	0.04	0.06	0.07	0.09	0.11	0.06	0.54	0.08	0.08	0.05	2.80
	71	308	0.00	0.01	0.02	0.03	0.04	0.04	0.05	0.07	0.09	0.13	0.54	0.06	0.06	0.04	2.52
	72[b]	219	0.00	0.00	0.02	0.03	0.04	0.06	0.08	0.11	0.13	0.15	0.41	0.08	0.08	0.05	3.10
Chicago, Ill. (141220002A10)	62	283	0.00	0.04	0.05	0.07	0.09	0.11	0.13	0.16	0.19	0.21	0.56	0.12	0.08	0.10	2.14
	63	315	0.00	0.04	0.06	0.08	0.09	0.11	0.12	0.13	0.16	0.20	0.38	0.11	0.06	0.10	1.99
	64	318	0.00	0.04	0.06	0.07	0.09	0.11	0.13	0.15	0.17	0.24	0.71	0.13	0.09	0.10	2.06
	65	317	0.00	0.04	0.06	0.08	0.10	0.12	0.13	0.15	0.17	0.21	0.43	0.12	0.07	0.11	1.98
	66	286	0.00	0.04	0.07	0.09	0.11	0.13	0.15	0.17	0.19	0.23	0.51	0.13	0.08	0.12	1.99
	67	311	0.00	0.03	0.05	0.07	0.08	0.10	0.12	0.13	0.15	0.18	0.43	0.10	0.06	0.09	2.16
	68	319	0.00	0.03	0.04	0.06	0.07	0.09	0.10	0.12	0.15	0.19	0.37	0.10	0.06	0.08	2.13
	69	318	0.01	0.04	0.06	0.09	0.10	0.13	0.15	0.18	0.21	0.26	1.02	0.15	0.11	0.12	2.03
	70	320	0.00	0.04	0.08	0.10	0.13	0.15	0.19	0.24	0.28	0.33	0.65	0.18	0.11	0.14	2.31
	71[b]	268	0.00	0.04	0.07	0.09	0.11	0.13	0.16	0.20	0.22	0.27	0.51	0.15	0.09	0.12	2.08
	72	321	0.01	0.04	0.07	0.10	0.13	0.15	0.18	0.21	0.24	0.29	0.53	0.16	0.09	0.13	2.10
New Orleans, La. (192020004A10)	62[b]	259	0.00	0.01	0.01	0.02	0.03	0.04	0.05	0.07	0.09	0.13	0.36	0.06	0.05	0.04	2.77
	63	325	0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.02	0.03	0.04	0.24	0.02	0.12	0.02	2.45
St. Louis, Mo. (264280002A10)	64[b]	259	0.00	0.01	0.02	0.04	0.04	0.06	0.07	0.08	0.09	0.12	0.44	0.06	0.04	0.05	2.22
	65	333	0.00	0.00	0.01	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.19	0.04	0.03	0.03	2.70
	66	294	0.00	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.12	0.49	0.05	0.05	0.04	2.90
	67	326	0.00	0.01	0.01	0.02	0.03	0.04	0.05	0.07	0.08	0.11	0.26	0.05	0.04	0.04	2.68
	68[b]	256	0.00	0.01	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.11	0.28	0.05	0.04	0.04	2.80
	69	322	0.00	0.00	0.01	0.02	0.03	0.04	0.05	0.07	0.09	0.12	0.51	0.05	0.06	0.03	3.10
	70[b]	228	0.00	0.01	0.02	0.02	0.04	0.06	0.08	0.11	0.14	0.17	0.47	0.08	0.07	0.05	2.92
	71	311	0.00	0.01	0.02	0.03	0.05	0.16	0.08	0.10	0.12	0.15	0.36	0.07	0.06	0.06	2.47
	72[b]	234	0.00	0.01	0.02	0.03	0.05	0.07	0.10	0.12	0.14	0.19	0.40	0.09	0.07	0.06	2.48
Cincinnati, Ohio (361220003A10)	62	286	0.00	0.01	0.01	0.02	0.02	0.03	0.04	0.06	0.08	0.12	0.42	0.05	0.05	0.03	2.74
	63[b]	273	0.00	0.01	0.01	0.02	0.02	0.03	0.04	0.05	0.08	0.12	0.26	0.05	0.04	0.03	2.52
	64	334	0.00	0.01	0.01	0.01	0.02	0.03	0.04	0.05	0.09	0.13	0.45	0.05	0.07	0.03	2.90
	65	310	0.00	0.00	0.01	0.01	0.02	0.02	0.03	0.05	0.07	0.11	0.43	0.04	0.05	0.03	2.86
	66[b]	206	0.00	0.01	0.01	0.12	0.03	0.04	0.05	0.06	0.09	0.14	0.67	0.06	0.07	0.04	2.73
	67[b]	188	0.00	0.01	0.02	0.02	0.02	0.03	0.04	0.05	0.08	0.13	0.53	0.06	0.08	0.04	2.62
	68[b]	136	0.00	0.01	0.03	0.04	0.05	0.07	0.10	0.14	0.18	0.29	0.69	0.12	0.14	0.07	3.03
	69[b]	196	0.00	0.00	0.01	0.02	0.03	0.04	0.05	0.07	0.10	0.18	0.56	0.07	0.09	0.04	3.04
	70[b]	129	0.00	0.00	0.01	0.02	0.03	0.03	0.04	0.05	0.08	0.13	0.52	0.05	0.07	0.03	2.96
	71[b]	215	0.00	0.01	0.02	0.03	0.03	0.04	0.05	0.07	0.10	0.20	0.48	0.07	0.07	0.05	2.64
	72[b]	251	0.00	0.01	0.02	0.02	0.03	0.04	0.05	0.07	0.09	0.17	0.47	0.06	0.07	0.04	2.64

TABLE 5-4 FREQUENCY DISTRIBUTION DATA FOR 6-TO 9-a.m. NITRIC OXIDE CONCENTRATIONS AT CAMP SITES, 1962-72[24] (cont'd).

(ppm)

Site	Year	Number of samples	Min.	Percentile[a]									Max.	Arithmetic		Geometric	
				10	20	30	40	50	60	70	80	90		Mean	Std dev.	Mean	Std dev.
Philadelphia, Pa.	62[b]	239	0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.02	0.03	0.06	0.23	0.02	0.02	0.02	2.54
	63[b]	233	0.00	0.00	0.01	0.02	0.03	0.04	0.05	0.07	0.10	0.15	0.79	0.07	0.10	0.04	3.30
	64	302	0.00	0.01	0.02	0.03	0.03	0.04	0.06	0.07	0.10	0.14	0.42	0.06	0.06	0.05	2.61
	65	290	0.00	0.01	0.02	0.02	0.04	0.04	0.06	0.07	0.10	0.16	0.64	0.07	0.07	0.05	2.65
	66	336	0.00	0.01	0.02	0.03	0.04	0.05	0.06	0.08	0.10	0.16	0.79	0.07	0.08	0.05	2.50
	67	296	0.00	0.02	0.03	0.04	0.05	0.07	0.08	0.10	0.13	0.17	0.71	0.09	0.08	0.07	2.38
	68[b]	266	0.00	0.01	0.02	0.03	0.04	0.05	0.07	0.08	0.12	0.17	0.55	0.08	0.08	0.05	2.60
	69	276	0.00	0.01	0.12	0.03	0.04	0.05	0.06	0.07	0.09	0.12	0.38	0.06	0.05	0.05	2.48
	70	305	0.00	0.01	0.03	0.04	0.05	0.06	0.08	0.10	0.12	0.16	0.81	0.08	0.09	0.06	2.47
	71	278	0.00	0.01	0.02	0.02	0.03	0.04	0.05	0.07	0.09	0.13	0.42	0.06	0.06	0.04	2.78

[a] Concentrations greater than or equal to specified value in indicated percentage of samples.

[b] Yearly standard exceeded.

TABLE 5-5. FREQUENCY DISTRIBUTION DATA FOR 6-TO 9-a.m. NITROGEN DIOXIDE CONCENTRATIONS AT CAMP SITES, 1962-72[24]

(ppm)

Site	Year	Number of samples	Min.	Percentile[a]									Max.	Arithmetic		Geometric	
				10	20	30	40	50	60	70	80	90		Mean	Std. dev.	Mean	Std. dev.
San Francisco, Calif. (056860002A10)	62[b]	142	0.00	0.00	0.00	0.01	0.01	0.01	0.02	0.03	0.03	0.04	0.09	0.02	0.01	0.02	2.23
	63[b]	155	0.00	0.01	0.02	0.02	0.03	0.03	0.04	0.04	0.04	0.06	0.19	0.03	0.02	0.03	1.80
	64[b]	10	0.01	0.01	0.02	0.03	0.04	0.04	0.04	0.04	0.05	0.05	0.06	0.04	0.01	0.04	1.54
Denver, Colo. (060580002A10)	65[b]	247	0.01	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.09	0.04	0.01	0.04	1.49
	66	324	0.00	0.02	0.03	0.03	0.03	0.04	0.04	0.04	0.05	0.05	0.10	0.04	0.01	0.04	1.47
	67[b]	180	0.00	0.02	0.02	0.03	0.03	0.04	0.04	0.04	0.05	0.06	0.09	0.04	0.01	0.04	1.57
	68[b]	226	0.00	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.11	0.04	0.01	0.04	1.64
	69	283	0.00	0.01	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.05	0.15	0.03	0.01	0.03	1.63
	70	282	0.00	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.05	0.07	0.13	0.04	0.02	0.04	1.76
	71[b]	171	0.00	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.06	0.10	0.03	0.01	0.03	1.66
	72[b]	253	0.00	0.01	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.07	0.12	0.04	0.02	0.04	1.78
Washington, D.C. (090020002A10)	62	313	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.16	0.03	0.01	0.03	1.76
	63	312	0.00	0.01	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.06	0.13	0.03	0.02	0.03	1.75
	64	308	0.01	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.06	0.15	0.03	0.01	0.04	1.63
	65	336	0.00	0.02	0.02	0.03	0.03	0.03	0.03	0.04	0.05	0.05	0.18	0.03	0.01	0.03	1.55
	66	275	0.00	0.02	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.05	0.13	0.03	0.01	0.03	1.61
	67	305	0.00	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.14	0.04	0.02	0.04	1.65
	68[b]	237	0.00	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.06	0.08	0.18	0.05	0.02	0.05	1.56
Washington, D.C. (090020003A10)	69[b]	254	0.00	0.02	0.02	0.03	0.03	0.04	0.04	0.04	0.05	0.06	0.11	0.04	0.01	0.04	1.56
	70	298	0.00	0.02	0.03	0.04	0.04	0.05	0.06	0.06	0.07	0.09	0.16	0.05	0.02	0.05	1.84
	71	318	0.01	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.04	0.05	0.11	0.03	0.01	0.04	1.50
	72[b]	214	0.00	0.00	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.09	0.40	0.05	0.06	0.04	2.55
Chicago, Ill. (141220002A10)	62	293	0.00	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.07	0.15	0.04	0.02	0.04	1.77
	63	318	0.00	0.02	0.03	0.03	0.03	0.03	0.04	0.04	0.05	0.06	0.11	0.04	0.01	0.04	1.49
	64	303	0.00	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.06	0.07	0.16	0.04	0.02	0.04	1.57
	65	330	0.01	0.02	0.03	0.03	0.03	0.04	0.04	0.04	0.05	0.06	0.11	0.04	0.01	0.04	1.43
	66	304	0.00	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.07	0.09	0.23	0.05	0.02	0.05	1.56
	67	290	0.00	0.02	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.07	0.17	0.04	0.02	0.04	1.62
	68	312	0.01	0.03	0.03	0.03	0.04	0.04	0.05	0.05	0.05	0.06	0.10	0.04	0.01	0.04	1.45
	69	330	0.00	0.03	0.03	0.03	0.04	0.04	0.05	0.05	0.06	0.07	0.17	0.04	0.02	0.05	1.51
	70	329	0.00	0.03	0.04	0.04	0.04	0.05	0.05	0.06	0.07	0.08	0.14	0.05	0.02	0.05	1.40
	71	285	0.02	0.03	0.03	0.04	0.04	0.04	0.05	0.06	0.07	0.08	0.39	0.06	0.05	0.05	1.67
	72	336	0.00	0.03	0.03	0.04	0.04	0.04	0.05	0.06	0.06	0.08	0.16	0.05	0.02	0.05	1.60
New Orleans, La. (192020004A10)	62	288	0.00	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.07	0.14	0.04	0.02	0.04	1.79
	63	289	0.00	0.00	0.01	0.01	0.01	0.01	0.02	0.02	0.02	0.03	0.05	0.01	0.00	0.02	1.86

TABLE 5-5. FREQUENCY DISTRIBUTION DATA FOR 6-TO 9-a.m. NITROGEN DIOXIDE CONCENTRATIONS AT CAMP SITES, 1962-72[24] (cont'd).

(ppm)

				Percentile[a]										Arithmetic		Geometric	
Site	Year	Number of samples	Min.	10	20	30	40	50	60	70	80	90	Max.	Mean	Std. dev.	Mean	Std. dev.
St. Louis, Mo.	64[b]	255	0.00	0.01	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.16	0.03	0.02	0.03	1.85
(264280002A10)	65	348	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.02	0.03	0.04	0.08	0.02	0.01	0.02	1.80
	66	322	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.04	0.04	0.06	0.12	0.03	0.01	0.03	1.80
	67	352	0.00	0.01	0.01	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.06	0.02	0.01	0.02	1.83
	68	322	0.00	0.00	0.01	0.01	0.02	0.02	0.02	0.02	0.03	0.04	0.11	0.02	0.01	0.02	1.94
	69[b]	258	0.00	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.04	0.05	0.11	0.02	0.01	0.02	2.11
	70[b]	233	0.00	0.01	0.02	0.02	0.02	0.02	0.03	0.03	0.04	0.05	0.10	0.02	0.01	0.03	1.66
	71	309	0.00	0.02	0.02	0.02	0.02	0.03	0.03	0.04	0.05	0.06	0.11	0.03	0.01	0.03	1.61
	72[b]	231	0.01	0.02	0.02	0.03	0.03	0.04	0.05	0.06	0.07	0.08	0.16	0.04	0.02	0.04	1.66
Cincinnati, Ohio	62	292	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.13	0.03	0.01	0.03	1.82
(36122000A10)	63	304	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.13	0.03	0.01	0.03	1.71
	64	336	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.06	0.15	0.03	0.01	0.03	1.77
	65	313	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.11	0.03	0.01	0.03	1.45
	66[b]	238	0.00	0.02	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.10	0.03	0.01	0.03	1.69
	67[b]	223	0.00	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.03	0.04	0.09	0.02	0.01	0.03	1.52
	68	280	0.00	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.09	0.03	0.01	0.03	1.59
	69[b]	231	0.00	0.02	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.10	0.03	0.01	0.03	1.56
	70[b]	129	0.01	0.02	0.03	0.03	0.03	0.03	0.04	0.04	0.05	0.07	0.12	0.04	0.02	0.04	1.53
	71[b]	236	0.00	0.01	0.02	0.02	0.02	0.03	0.04	0.04	0.05	0.07	0.11	0.03	0.02	0.03	2.00
	72[b]	227	0.00	0.02	0.03	0.03	0.04	0.04	0.04	0.05	0.05	0.06	0.13	0.04	0.01	0.04	1.64
Philadelphia, Pa.	62[b]	211	0.00	0.00	0.01	0.01	0.02	0.02	0.02	0.03	0.03	0.04	0.10	0.02	0.01	0.02	1.56
(397140002A10)	63	286	0.00	0.01	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.07	0.15	0.03	0.02	0.03	1.97
	64	281	0.00	0.02	0.02	0.03	0.03	0.03	0.04	0.04	0.05	0.07	0.20	0.04	0.02	0.04	1.72
	65	286	0.00	0.02	0.02	0.02	0.03	0.03	0.03	0.04	0.05	0.05	0.12	0.03	0.01	0.03	1.58
	66	340	0.00	0.02	0.02	0.02	0.03	0.03	0.04	0.04	0.05	0.06	0.11	0.03	0.01	0.03	1.66

[a] Concentrations greater than or equal to specified value in indicated percentage of samples.

[b] Yearly standard exceeded.

by far the most important source of hydrocarbon and nitrogen oxide emissions. In addition, hydrocarbon and nonhydrocarbon organic emissions also arise from the use of such organics in the processing of raw materials.

From a pollution control standpoint, the distinction made between natural and manmade sources and the use that is made of annual emission rate data are not entirely satisfactory. For example, anthropogenic emissions such as those from leaking fuel lines, home appliances, etc. cannot be subjected to systematic control because of their accidental nature. Thus from a control standpoint, such emissions may be considered to be natural. To generalize, classification of sources and emissions as controllable and uncontrollable may be more appropriate than the distinction now in use. Also, emission rate data reflecting annual or even daily averages are inadequate in that they mask seasonal and diurnal variations in emission rates. Considering that only the morning-to-noon emissions in the summer-to-fall season are of main photochemical consequence, it is evident that ignoring the seasonal and diurnal variations in emission rates does not permit equitable assessment of the various emission sources.

The sections that follow deal with the main anthropogenic and natural emissions; emission rate data are presented in the form in which they are available—mostly as daily and yearly averages.

TABLE 5-6. NITRIC OXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963-67[2]

(ppm)

Place, site no., averaging time	Maximum for year					Percentile[b]							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Anaheim-176:													
1 hr	0.29	0.30	0.70	0.40	0.66	0.62	0.40	0.22	0.07	0.03	0.01	0.00	0.00
8 hr	0.18	0.19	0.29	0.24	0.41	—	0.28	0.18	0.07	0.03	0.02	0.01	0.00
1 day	0.11	0.09	0.17	0.15	0.18	—	—	0.12	0.06	0.03	0.02	0.01	0.00
1 mo	0.05	0.04	0.05	0.04	0.07	—	—	—	0.06	0.04	0.02	0.02	0.01
1 yr	0.02	—	—	0.02	0.04	—	—	—	—	—	0.02	—	—
Oakland-327:													
1 hr	0.93	0.93	0.66	0.68	0.91	0.92	0.70	0.41	0.15	0.05	0.02	0.01	0.00
8 hr	0.57	0.60	0.34	0.34	0.52	—	0.55	0.32	0.14	0.05	0.03	0.01	0.00
1 day	0.33	0.35	0.26	0.26	0.30	—	—	0.26	0.13	0.06	0.03	0.02	0.01
1 mo	0.02	0.14	0.11	0.13	0.11	—	—	—	0.11	0.07	0.04	0.02	0.02
1 yr	—	0.07	0.05	0.05	0.05	—	—	—	—	—	0.05	—	—
Riverside-126:													
1 hr	—	1.10	0.57	0.43	0.52	0.74	0.47	0.29	0.11	0.04	0.02	0.01	0.00
8 hr	—	0.59	0.36	0.26	0.26	—	0.38	0.21	0.10	0.04	0.02	0.01	0.00
1 day	—	0.11	0.10	0.08	0.08	—	—	0.18	0.09	0.05	0.03	0.01	0.00
1 yr	—	—	0.04	0.04	0.04	—	—	—	—	—	0.04	—	—
Sacramento-276:													
1 hr	1.08	1.08	0.97	0.75	0.90	0.97	0.70	0.37	0.09	0.03	0.02	0.00	0.00
8 hr	0.60	0.53	0.62	0.49	0.58	—	0.54	0.29	0.09	0.03	0.02	0.01	0.00
1 day	0.35	0.26	0.28	0.23	0.33	—	—	0.22	0.09	0.04	0.02	0.01	0.00
1 mo	0.14	0.09	0.10	0.07	0.07	—	—	—	0.07	0.05	0.03	0.01	0.01
1 yr	0.04	0.04	0.03	0.04	0.03	—	—	—	—	—	0.04	—	—
San Bernardino-151:													
1 hr	0.25	0.25	0.34	0.50	0.36	0.47	0.32	0.15	0.05	0.02	0.01	0.00	0.00
8 hr	0.12	0.12	0.16	0.26	0.15	—	0.25	0.12	0.05	0.02	0.01	0.01	0.00
1 day	0.06	0.06	0.10	0.20	0.11	—	—	0.09	0.05	0.03	0.02	0.01	0.00
1 mo	0.03	0.02	0.04	0.07	0.05	—	—	—	0.04	0.03	0.02	0.01	0.00
1 yr	0.01	—	—	—	0.03	—	—	—	—	—	0.03	—	—
San Diego-101:													
1 hr	0.74	1.10	0.90	1.20	0.80	1.00	0.65	0.38	0.10	0.02	0.01	0.00	0.00
8 hr	0.45	0.63	0.43	0.42	0.34	—	0.42	0.26	0.10	0.03	0.01	0.00	0.00
1 day	0.24	0.21	0.23	0.26	0.22	—	—	0.20	0.10	0.04	0.01	0.00	0.00
1 mo	0.14	0.08	0.09	0.12	0.10	—	—	—	0.09	0.05	0.02	0.01	0.00
1 yr	0.04	0.02	0.03	0.05	0.04	—	—	—	—	—	0.04	—	—
Stockton-252:													
1 hr	0.38	0.50	0.48	0.87	0.36	0.68	0.47	0.27	0.08	0.02	0.01	0.00	0.00
8 hr	0.18	0.29	0.33	0.50	0.16	—	0.39	0.23	0.07	0.02	0.01	0.00	0.00
1 day	0.14	0.15	0.23	0.26	0.09	—	—	0.19	0.07	0.03	0.01	0.01	0.00
1 mo	0.04	0.06	0.11	0.11	0.02	—	—	—	0.06	0.03	0.02	0.01	0.00
1 yr	—	0.03	0.03	0.03	0.01	—	—	—	—	—	0.03	—	—

[a] Determined by continuous Griess-Saltzman method.

[b] Concentrations greater than or equal to specified value in indicated percentage of samples.

TABLE 5-7. NITROGEN DIOXIDE CONCENTRATION[a] IN CALIFORNIA BY AVERAGING TIME AND FREQUENCY, 1963-67

(ppm)

Place, site no., averaging time	Maximum for year					Percentile[b]							
	63	64	65	66	67	0.01	0.1	1	10	30	50	70	90
Anaheim-176:													
1 hr	0.20	0.22	0.23	0.27	0.27	0.25	0.21	0.15	0.07	0.05	0.03	0.02	0.01
8 hr	0.15	0.13	0.16	0.15	0.19	—	0.18	0.13	0.07	0.04	0.03	0.02	0.01
1 day	0.11	0.11	0.13	0.12	0.13	—	—	0.10	0.06	0.04	0.03	0.02	0.01
1 mo	0.06	0.04	0.05	0.05	0.07	—	—	—	0.06	0.05	0.04	0.03	0.02
1 yr	0.03	—	—	0.04	0.04	—	—	—	—	—	0.04	—	—
Oakland-327:													
1 hr	0.28	0.41	0.23	0.29	0.33	0.33	0.23	0.14	0.07	0.04	0.03	0.02	0.01
8 hr	0.19	0.25	0.15	0.18	0.23	—	0.20	0.13	0.06	0.04	0.03	0.02	0.01
1 day	0.10	0.16	0.10	0.13	0.15	—	—	0.10	0.06	0.04	0.03	0.02	0.02
1 mo	0.05	0.05	0.06	0.05	0.06	—	—	—	0.05	0.04	0.03	0.03	0.02
1 yr	0.03	0.03	0.03	0.03	0.04	—	—	—	—	—	0.03	—	—
Riverside-126:													
1 hr	0.27	0.56	0.49	0.25	0.31	0.49	0.32	0.18	0.09	0.05	0.03	0.02	0.01
8 hr	0.18	0.26	0.31	0.18	0.23	—	0.29	0.15	0.08	0.05	0.04	0.02	0.01
1 day	0.14	0.19	0.25	0.13	0.17	—	—	0.14	0.07	0.05	0.04	0.03	0.02
1 mo	0.06	0.05	0.08	0.05	0.05	—	—	—	0.05	0.05	0.04	0.04	0.03
1 yr	—	0.04	0.05	0.04	0.04	—	—	—	—	—	0.04	—	—
Sacramento-276:													
1 hr	0.29	0.32	0.30	0.27	0.30	0.30	0.22	0.14	0.07	0.04	0.03	0.02	0.01
8 hr	0.17	0.21	0.20	0.21	0.20	—	0.19	0.12	0.06	0.04	0.03	0.02	0.01
1 day	0.13	0.13	0.13	0.15	0.13	—	—	0.11	0.06	0.04	0.03	0.02	0.02
1 mo	0.07	0.07	0.07	0.05	0.05	—	—	—	0.05	0.04	0.03	0.03	0.02
1 yr	0.04	0.04	0.04	0.03	0.04	—	—	—	—	—	0.04	—	—
San Bernardino-151:													
1 hr	0.14	0.06	0.11	0.25	0.22	0.23	0.19	0.13	0.07	0.04	0.02	0.01	0.00
8 hr	0.12	0.03	0.07	0.14	0.17	—	0.16	0.12	0.06	0.04	0.02	0.01	0.00
1 day	0.06	0.02	0.05	0.11	0.13	—	—	0.10	0.06	0.04	0.03	0.01	0.00
1 mo	0.03	—	0.03	0.06	0.07	—	—	—	0.05	0.04	0.03	0.01	0.00
1 yr	0.01	—	—	0.04	0.05	—	—	—	—	—	0.04	—	—
San Diego-101:													
1 hr	0.33	0.35	0.52	0.40	0.34	0.35	0.22	0.12	0.06	0.03	0.01	0.00	0.00
8 hr	0.18	0.15	0.22	0.19	0.17	—	0.18	0.11	0.06	0.03	0.02	0.01	0.00
1 day	0.12	0.09	0.12	0.12	0.08	—	—	0.09	0.05	0.03	0.02	0.01	0.00
1 mo	0.06	0.06	0.04	0.06	0.03	—	—	—	0.04	0.03	0.02	0.01	0.01
1 yr	0.03	0.02	0.02	0.03	0.02	—	—	—	—	—	0.02	—	—
Stockton-252:													
1 hr	0.13	0.22	0.14	0.16	0.18	0.20	0.16	0.09	0.04	0.02	0.02	0.01	0.01
8 hr	0.08	0.15	0.11	0.11	0.10	—	0.12	0.07	0.04	0.03	0.02	0.01	0.01
1 day	0.06	0.08	0.08	0.07	0.06	—	—	0.06	0.04	0.03	0.02	0.02	0.01
1 mo	0.02	0.05	0.03	0.03	0.03	—	—	—	0.03	0.02	0.02	0.02	0.01
1 yr	—	0.03	0.02	0.02	0.02	—	—	—	—	—	0.02	—	—

[a] Determined by continuous Griess-Saltzman method.

[b] Concentration greater than or equal to specified value in indicated percentage of samples.

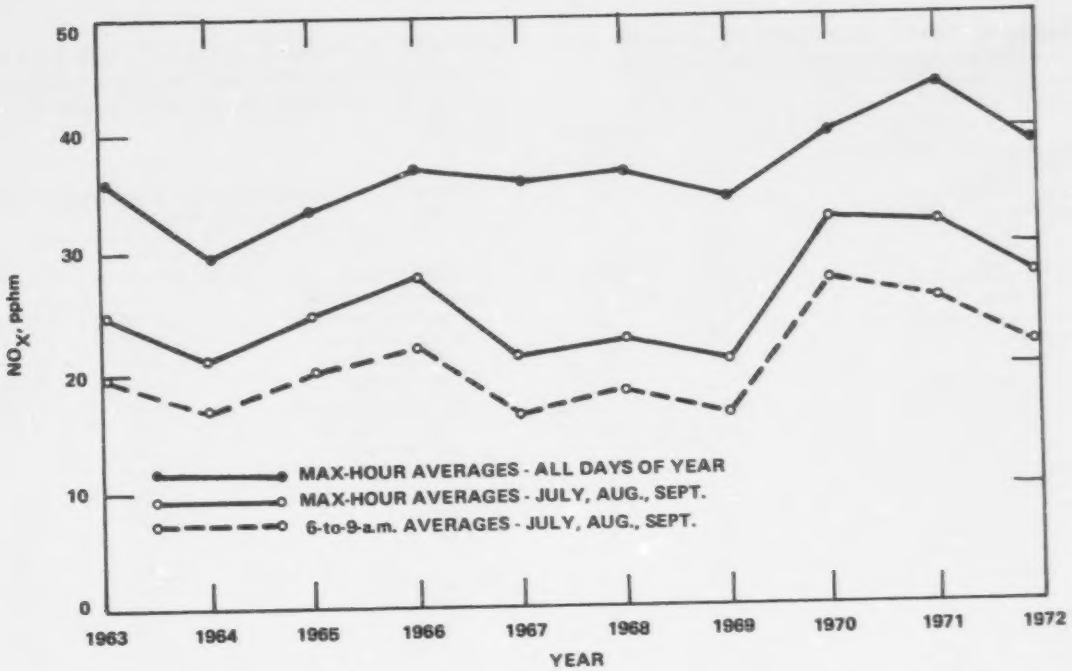

Figure 5-4. Oxides of nitrogen trends in Los Angeles, 1963- 1972, 6-to-9-a.m. and maximum 1-hr average concentrations.[16]

Summary of Organic Emissions Data

A summary of several estimates of the total hydrocarbon emissions from mobile, stationary, and natural sources within the United States is presented in Tables 5-8 and 5-9.[84] The differences between estimates are due primarily to differences in calculation techniques, data sources, and underlying assumptions. Most investigators agree that emission estimates are improving as more is learned about sources and measurement techniques, and that the more recent estimates in Tables 5-8 and 5-9 are probably more reliable than earlier ones.

TABLE 5-9. TOTAL YEARLY HYDROCARBON EMISSION RATE FOR THE CONTINENTAL UNITED STATES, BASED ON LEAF BIOMASS[84]

Item	Emissions, 10^6 tons/yr
Summer (5 months)	52.4
Vegetation emissions	48.2
Leaf litter emissions	4.2
Winter (7 months)	34.9
Vegetation emissions	34.9
Leaf litter emissions	~0
Total yearly emissions from vegetation and leaf litter	87.3
Total year emissions from anthropogenic sources	21.2
Natural emissions as percent of total emissions from all sources	80%

TABLE 5-8. ESTIMATES OF TOTAL HYDROCARBON EMISSIONS FROM MANMADE AND NATURAL SOURCES

	Estimate, 10^6 metric tons/yr					
Source type	1972 Rasmussen	1972 MSA[85] Research Corp.	1972[63] NEDS	1972[86] NADB	1973[63] NADB	1974[63] NADB
Mobile	ND	ND	14.8	12.8	12.5	11.6
Stationary	ND	23.1	10.2	15.1	15.5	15.5
Natural	2.3[a]	ND	ND	ND	ND	ND

[a]Emissions from tree foliage only (see J. Air Poll. Contr. Assoc. 22:537-543, 1972)

More detailed hydrocarbon emission inventories for the United States and hydrocarbon emission trends in 1970-75 are presented in Tables 5-10 and 5-11.[63] Information presented in Tables 5-10 and 5-11 was taken from the EPA Data File of Nationwide Emissions[63,86] and has been validated recently. The data in Tables 5-10 and 5-11 should not be compared with previously published data on 1970-75 emissions.

TABLE 5-10. 1974 NATIONWIDE ESTIMATES OF TOTAL HYDROCARBON SOURCES AND EMISSIONS[66]

Source category	Emissions, 10^6 metric tons/yr 1974	Emissions, 10^6 metric tons/yr 1975 (preliminary)
Transportation (total)	(11.3)	(10.6)
Highway	9.8	9.1
Non-highway	1.5	1.5
Stationary fuel combustion (total)	(1.6)	(1.3)
Electric utilities	0.1	0.1
Other	1.5	1.2
Industrial processes (total)	(3.3)	(3.2)
Chemicals	1.6	1.5
Petroleum refining	0.8	0.8
Metals	0.2	0.2
Others	0.7	0.7
Solid waste (total)	(.9)	(.8)
Miscellaneous (total)	(12.7)	(12.2)
Forest wildfires	0.5	0.5
Forest-managed burning	0.2	0.2
Agricultural burning	0.1	0.1
Coal refuse burning	0.1	0.1
Structural fires	0.0	<0.1
Organic solvents	8.1	7.5
Oil and gas production and marketing	3.7	3.8
Total	29.8	28.0

TABLE 5-11. NATIONWIDE TOTAL HYDROCARBON EMISSION TRENDS, 1970-75[66]

Year	Emission, 10^6 metric tons/yr
1970	30.7
1971	30.2
1972	30.9
1973	30.8
1974	29.8
1975	28.0

Table 5-12 presents a more detailed breakdown of hydrocarbon emissions from mobile sources than is given in either Table 5-10 or 5-11. Data for this table were obtained from the EPA Data File of Nationwide Emissions.[66] The table indicates the relatively large contribution that gasoline-fueled light vehicles make to total hydrocarbon emissions from mobile sources. Of course, it must be recognized that hydrocarbon emissions from light-duty, gasoline-fueled vehicles have been reduced since 1974, and the exhaust emission rate has decreased by about 70 percent.[65] Evaporative and crankcase emissions have remained essentially unchanged during this period. It is difficult to estimate national emission levels because of the uncertainties associated with vehicle populations, miles driven, and deterioration of emission control systems. But from the model developed by Heywood and Martin, a net decrease of about 28 percent has been projected for aggregate hydrocarbon emissions from automobiles during the 1972-77 period.[38] Tables 5-13 and 5-14 give organic emission and reactive-organic emission inventories for the Los Angeles Air Quality Control Region.[81]

TABLE 5-12. HYDROCARBON EMISSIONS FROM MOBILE SOURCES[63]

Source category	Emissions, 10^6 metric tons/yr	% of total
Land vehicles (total):		
Gasoline-fueled (total)	(10.11)	(89.23)
Light vehicles	8.41	74.23
Heavy vehicles	1.26	11.12
Off-highway	0.44	3.88
Diesel-fueled (total)	(0.46)	(4.06)
Heavy vehicles	0.15	1.32
Off-highway	0.13	1.15
Rail	0.18	1.59
Aircraft (total)	(0.33)	(2.91)
Military	0.22	1.94
Civil	0.04	0.35
Commercial	0.07	0.62
Vessels (total)	(0.43)	(3.79)
Coal	<0.01	<0.01
Diesel	0.02	0.18
Gasoline	0.41	3.61
Total	11.33	—

Hydrocarbon Emissions from Natural Sources

The major natural sources that have been identified and for which quantitative estimates are available are the biologic decomposition of organic matter, seepage from natural gas and oil fields, and emission of volatile compounds from plants. However, there is information in the literature that indicates that there are many other natural sources of hydrocarbons and oxygenates that have not been considered heretofore. This section discusses the major natural sources for which quantitative estimates are available and indicates some of the minor sources that have been identified.

Methane is produced in the anaerobic bacterial decomposition of organic matter in swamps, lakes, marshes, paddy fields, etc. Koyama[46] has estimated the global production of methane as 2.7 x 10^{14} g/year (300 million tons/year) on the basis of his measurements of methane fermentation in various soils and lake sediments under controlled experimental conditions. Robinson and Robbins[74] have estimated the production from swamps and humid tropical areas and added it to Koyama's figure to derive an estimate of methane production of 14.5 x 10^{14} g/year (1.6 billion tons/year). Other sources of methane are gas, oil, and coal fields.[5] The seepage of natural gas from these areas has been determined by Ehhalt,[33] through carbon-14 measurements, to contribute as much as 25 percent to the total atmospheric methane. The remaining 75 percent is of recent biogenic origin. Our understanding of the natural sources of methane is still highly limited. As a result, current estimates of global production remain speculative.

Plants release a variety of volatile organic substances, including ethylene, isoprene, α-pinene, and a variety of other terpenes. Rasmussen[68] and Rasmussen and Went[70] measured and reported ambient concentrations of volatile plant organics (such as isoprene, α- and β-pinene, limonene, and myrcene) in air at remote sites. From the average concentration of 10 ppb that they measured, a global production rate of volatile plant organics was estimated to be 438 million tons/year. From the available estimates of methane and terpene emissions, it is estimated that worldwide natural hydrocarbon emissions are

TABLE 5-13. ORGANIC EMISSION INVENTORY FOR THE METROPOLITAN LOS ANGELES AIR QUALITY CONTROL REGION, 1972[81]

	Weight emissions		Molar emissions		
Source category	Tons/day	Weight % of total	10^{-2} ton moles/day	Mole % of total	Average molecular weight
Stationary sources: Organic fuels and combustion					
Petroleum production and refining:					
Petroleum production	62	2.3	214	5.9	29
Petroleum refining	50	1.9	54	1.5	93
Gasoline marketing:					
Underground service station tanks	48	1.8	83	2.3	58
Auto tank filling	104	4.0	141	3.9	74
Fuel combustion	23	0.9	92	2.5	25
Waste burning and fires	41	1.6	124	3.4	33
Stationary sources: Organic chemicals					
Surface coating:					
Heat treated	14	0.5	17	0.5	82
Air dried	129	5.0	148	4.1	87
Dry cleaning:					
Petroleum-based solvent	16	0.6	13	0.4	126
Synthetic solvent (PCE)	25	1.0	15	0.4	166
Degreasing:					
TCE solvent	11	0.4	8	0.2	132
1,1,1-T solvent	95	3.6	71	2.0	134
Printing:					
Rotogravure	31	1.2	38	1.0	82
Flexigraphic	15	0.6	26	0.7	57
Industrial process sources:					
Rubber and plastic manufacturing	42	1.6	58	1.6	73
Pharmaceutical manufacturing	16	0.6	21	0.6	75
Miscellaneous operations	83	3.2	104	2.9	80
Mobile sources:					
Gasoline-powered vehicles:					
Light duty vehicles:					
Exhaust emissions	780	30.0	1130	31.2	69
Evaporative emissions	481	18.5	529	14.6	91
Heavy duty vehicles:					
Exhaust emissions	285	10.9	413	11.4	69
Evaporative emissions	67	2.6	74	2.0	91
Other gasoline-powered equipment:					
Exhaust emissions	110	4.2	159	4.4	69
Evaporative emissions	22	0.8	24	0.7	91
Diesel-powered motor vehicles	12	0.5	13	0.4	89
Aircraft:					
Jet	20	0.8	17	0.5	121
Piston	22	0.9	39	1.1	56
Total of weighted average	2604	100	3625	100	71.9

about 2 billion tons/year. However, there is evidence that many other volatile organics are emitted from natural sources into the atmosphere. These may be only trace amounts emitted on a local scale, but the total worldwide production can be very large.

Annual hydrocarbon emissions from natural sources in the United States can be estimated from the preceding information. If the natural emission of methane is uniform in all land areas, Robinson and Robbins'[74] estimate of the worldwide emission of methane (1.6 billion tons/year) can be used to calculate a U.S. emission of 100 million tons/year. The assumption that natural methane is emitted

TABLE 5-14. REACTIVE EMISSION INVENTORIES FOR THE METROPOLITAN LOS ANGELES AIR QUALITY CONTROL REGION[81]

Source category	Total emissions		Reactive emissions					
			Reactive tons/day[a]			% of total		
	Tons/day	% of total	2-group[b] scheme	5-group[b] scheme	6-group[b] scheme	2-group scheme	5-group scheme	6-group scheme
Stationary sources: Organic fuels and combustion:								
Petroleum production and refining:								
Petroleum production	62	2.3	24	28	18	1.4	1.7	1.1
Petroleum refining	50	1.9	33	27	27	1.9	1.6	1.6
Gasoline marketing:								
Underground service station tanks	48	1.8	47	40	40	2.7	2.4	2.4
Auto tank filling	104	4.0	94	76	77	5.4	4.6	4.7
Fuel combustion	23	0.9	6	13	8	0.3	0.8	0.5
Waste burning and fires	41	1.6	22	32	27	1.3	1.9	1.6
Stationary sources: Organic chemicals:								
Surface coating:								
Heat treated	14	0.5	9	8	8	0.5	0.5	0.5
Air dried	129	5.0	88	71	71	5.0	4.3	4.3
Dry cleaning:								
Petroleum based solvent	16	0.6	9	6	6	0.5	0.4	0.4
Synthetic solvent (PCE)	25	1.0	0	1	1	0.0	0.1	0.1
Degreasing:								
TCE solvent	11	0.4	6	5	5	0.3	0.3	0.3
1,1,1-T solvent	95	3.6	0	5	5	0.0	0.3	0.3
Printing:								
Rotogravure	31	1.2	21	16	16	1.2	1.0	1.0
Flexigraphic	15	0.6	15	14	14	0.8	0.8	0.8
Industrial process sources:								
Rubber and plastic manufacturing	42	1.6	33	39	39	1.9	2.3	2.4
Pharmaceutical manufacturing	16	0.6	10	9	9	0.6	0.5	0.5
Miscellaneous operations	83	3.2	40	38	38	2.3	2.5	2.3
Mobile sources:								
Gasoline-powered vehicles:								
Light duty vehicles:								
Exhaust emissions	780	30.0	562	562	562	32.1	33.9	34.2
Evaporative emissions	481	18.5	362	293	293	19.8	17.7	17.9
Heavy duty vehicles:								
Exhaust emissions	285	10.9	205	205	205	11.7	12.3	12.5
Evaporative emissions	67	2.6	48	41	41	2.7	2.5	2.5
Other gasoline-powered equipment:								
Exhaust emissions	110	4.2	79	79	79	4.5	4.8	4.8
Evaporative emissions	22	0.8	16	13	13	0.9	0.8	0.8
Diesel-powered motor vehicles	12	0.5	8	9	9	0.5	0.5	0.5
Aircraft:								
Jet	20	0.8	10	10	10	0.6	0.6	0.6
Piston	22	0.9	18	20	20	1.0	1.2	1.2
Total	2604	100	1749	1660	1641	100	100	100

[a] To convert to reactive ton moles per day, multiply by 0.0145.

[b] For reactivity definition, see reference 42.

uniformly from all land areas is probably in error, since emission rates are higher in tropical than in nontropical areas. More realistically, assuming that the U.S. methane emission rate per unit of land area is half the world average, the U.S. natural methane emission would be 50 million tons/year. Similarly, if the estimate of Rasmussen and Went[70] of worldwide terpene emission (438 million tons/year) is based on uniform distribution over the forested areas of the world, the U.S. emission would be 22 million tons/year. Emission of ethylene from plants in the United States has been estimated at 20,000 tons/year.[1] By combining these estimates, a natural hydrocarbon emission in the United States of about 72 million tons/year is obtained.

Hydrocarbon Emissions from Anthropogenic Sources

MOBILE SOURCES

As indicated in Table 5-13, light-duty, gasoline-fueled motor vehicles account for a vast majority of the hydrocarbons emitted into the environment from mobile sources. There are three primary sources of hydrocarbon emissions from the motor vehicle: crankcase ventilation, gasoline evaporation, and combustion exhaust. Crankcase blowby emissions were essentially eliminated by means of the positive crankcase ventilation system introduced in 1963; evaporative emissions were reduced by about 30 percent by means of adsorption-regeneration systems introduced in 1971.[65] Exhaust hydrocarbon emissions have been reduced by about 90 percent in new vehicles since 1968 with a variety of engine modifications, and most recently with the application of oxidation-catalyst systems. Although the true reductions in emissions are somewhat less than these figures because of control device deterioration, it is reasonable, nevertheless, to believe that the current and the more advanced controls of the future will effect a further reduction in motor vehicle aggregate hydrocarbon emissions through the middle to late 1980's.[38] Vehicle population growth and miles traveled will become the controlling factors subsequent to that time.

The patterns of hydrocarbon emissions from motor vehicles, as well as their mass, are dependent on a large number of factors. Driving patterns, emission control system deterioration, ambient temperature, pressure, humidity, and the type of fuel used are all significant factors.[11,58,87] The relative contributions of evaporative gasoline emissions and combustion exhaust in the aggregate are also important. Therefore, it is very difficult to speak in absolute terms about the relative abundances of hydrocarbon types in vehicle emissions. However, trends in emissions can be discussed with reasonable authority.

Historically, combustion exhaust dominated the hydrocarbon emissions from automobiles, and its pattern was typical of atmospheric hydrocarbons from motor vehicle sources. The advances in emission controls, however, have changed this situation. The automobile manufacturers have been very successful in controlling exhaust hydrocarbons. Since the 1975 model year, evaporative hydrocarbons have constituted the major fraction of the hydrocarbon aggregate from new automobiles.[65] It has been estimated that for a typical 1975 passenger car, evaporative gasoline accounts for about 60 percent of the total aggregate emissions, and combustion exhaust, 40 percent.[65]

The pattern of exhaust hydrocarbons has changed with the use of oxidation catalysts. Most catalytic control systems show greater activity for unsaturated hydrocarbons than saturated. They show very little activity for methane.[13,14] Thus the relative abundance of paraffinic hydrocarbons in the exhaust has increased with recent generation automobiles. Typically, exhaust from a catalyst-equipped automobile would contain about 62 percent paraffinic hydrocarbon, 17 percent aromatic hydrocarbon, 18 percent olefinic hydrocarbon, and 3 percent acetylenic hydrocarbon, as compared with 40, 24, 26 and 11 percent, respectively, for noncatalytically equipped automobiles. The methane levels generally range from about 10 to 30 percent. Evaporative gasoline is also dominated by paraffinic hydrocarbons, with the C_4-C_6 paraffins typically accounting for about 70 percent of the total.[55,83]

Thus, the trend in hydrocarbon emissions from passenger cars is toward an increased relative abundance of paraffinic hydrocarbons.[12] The oxidation-catalyst exhaust emission control device has generally resulted in an increased relative abundance of methane and a decreased relative abundance of unsaturated hydrocarbons, particularly notable in the olefinic and acetylenic hydrocarbons. Evaporative gasoline has become a more significant fraction of the total aggregate of motor vehicle hydrocarbon emissions.

Besides hydrocarbons, exhaust gases contain oxygenated hydrocarbon compounds such as aldehydes, ketones, alcohols, ethers, esters, acids, and phenols. The total oxygenate concentration is about 5 to 10 percent of the total hydrocarbon concentration. Aldehydes are generally believed to be the most important class of exhaust oxygenates.

A reasonably complete quantitative analysis of exhaust aldehydes is possible with gaschromatographic techniques. Formaldehyde is by far the predominant aldehyde, constituting about 60 to 70 percent of the total (on a volume basis); acetaldehyde is next, at about 10 percent; and propionaldehyde, acrolein, benzaldehyde, and the tolualdehydes are all found in appreciable amounts. As might be expected, the nature of the gasoline influences the aldehydes formed.[12,34,64,88,89]

There is little published information on noncarbonyl oxygenates such as ethers, alcohols, epoxides, and peroxides, but Seizinger and Dimitriades[75,76] measured 10 aldehydes, 6 ketones, and 16 noncarbonyl oxygenates in exhaust from 22 different simple fuels, each containing 1, 2, or 3 hydrocarbons. They developed formulas from their data that can be used to calculate the estimated concentrations of oxygenate in gasoline exhaust.

The composition of organic emissions from the other mobile sources (diesels, aircraft, etc.) is not well defined. Diesel exhaust organics consist of a light fraction, in the C_1-C_4 range, and a heavy fraction (C_8-C_{22}), with the relative amounts of the two fractions varying with engine load and speed (see Figure 5-1).[39] Several diesel exhaust organics have been identified in the course of researching diesel exhaust odor.[18] Such organics include indans, indenes, naphthalenes, and tetralins, as well as high molecular weight carbonyl and other oxygenated compounds. Exhaust organics from aircraft consist primarily of organics in the C_8-C_{22} range (see Figure 5-2).[79]

There is little doubt that organic emissions from gasoline-powered mobile sources are oxidant producers almost in their totality. Emissions from diesels and aircraft will participate in gas-phase reactions only partly. A substantial part of such emissions is expected to condense on surfaces, thus contributing significantly, perhaps, to visibility reduction.

STATIONARY SOURCES

A detailed description of the various stationary sources of organic emissions, including emission rate data, can be found in a recent National Academy of Sciences report.[61] Major constituents of the emissions associated with fuel combustion are organic acids, followed by hydrocarbons and aldehydes.[61] Such composition is entirely different from the combustion-related emissions from mobile sources in which hydrocarbons are the major constituent and organic acids are negligible. Industrial processes discharge a variety of organic compounds in the atmosphere, including C_4-C_6 hydrocarbons from refineries and aromatic hydrocarbons and acid derivatives, aldehydes, alcohols, and phenols from chemical processing operations. Solvent evaporation from painting, coating, drycleaning, printing, etc., result in substantial levels of emissions consisting mainly of petroleum naphtha. Forest fires, structural fires, and agricultural burning result in emissions that are difficult to characterize and nearly impossible to measure. Finally, gasoline marketing operations, (i.e. storage, transportation, and service station handling of gasoline) result in emissions consisting primarily of C_4-C_6 hydrocarbons and secondarily of whole gasoline vapors. The relative importance of the stationary source emissions, in terms both of amounts and oxidant-producing potential, is illustrated by the Los Angeles data shown in Tables 5-13 and 5-14.[81]

Emissions of Nitrogen Oxides

The distribution of nitrogen oxide (NO_x) emissions by major source categories is indicated in Table 5-15.[63] Data shown in this table have been validated recently and are therefore more reliable than previously reported data on 1974 NO_x emissions. Fuel combustion is the major cause of technology-associated emissions. In 1974, combustion of coal, oil, natural gas, and motor-vehicle fuel accounted for more than 22 of an estimated 23 million tons of manmade NO_x in the United States. An estimated 9.6 million tons was emitted from transportation sources, 7.4 million tons of which was from motor vehicles. Industrial processes, solid waste disposal, and other miscellaneous sources accounted for about 1 million tons of NO_x.

Relatively small quantities of NO_x are emitted from noncombustion industrial processes, mainly the manufacturing and use of nitric acid.[52] Even though total quantities may be small, high concentrations of NO_x can be emitted from some of these chemical processes. Electroplating, engraving, welding, metal cleaning, and explosive detonation also can be responsible for industrial NO_x emissions, as can the maufacture and use of liquid-NO_2-based rocket propellants.

TABLE 5-15. 1974 NATIONWIDE ESTIMATES OF NITROGEN OXIDE SOURCES AND EMISSIONS[63]

Source category	Emission, 10^6 metric tons/yr
Transportation (total)	(9.6)
Highway	7.4
Nonhighway	2.2
Stationary fuel combustion (total)	(12.1)
Electric utilities	6.3
Other	5.8
Industrial processes (total)	(0.6)
Chemicals	0.3
Petroleum refining	0.3
Other	<0.1
Solid waste (total)	(0.2)
Miscellaneous	(0.2)
Forest wildfires	0.1
Forest-managed burning	<0.1
Agricultural burning	<0.1
Coal refuse burning	0.1
Structural fires	<0.1
Total	22.7

Natural sources of nitrogen oxide emissions include biological processes in soil, atmospheric oxidation of ammonia (NH_3), and, possibly, photolysis of NO_2. Estimates of NO_x emission rates from natural sources vary considerably among investigators. Robinson and Robbins[73] estimated the annual NO_x emissions from biological processes to be 770 million metric tons (as NO_2), compared to 52 million metric tons from manmade sources. Other investigators question the importance of the biological processes, and they offer the oxidation of NH_3 resulting in 230 million tons of NO_x per year, as the main source.[54] As in the case of the hydrocarbon emissions, the natural sources of NO_x seem to dominate the anthropogenic ones. But again, as in the hydrocarbon case, this dominance has no significant relevance to the ambient oxidant problem or even to the ambient NO_2 problem, since the natural and anthropogenic emissions are, for the most part, segregated geographically, with the anthropogenic ones concentrated in the populated areas.

REACTIVITY OF ORGANIC EMISSIONS

From an air pollution standpoint, the photochemical reactivity of an organic pollutant denotes the intrinsic ability of the pollutant to participate in atmospheric chemical reactions that result in photochemical smog formation. The concept of hydrocarbon reactivity (the term "hydrocarbon" is meant here to encompass all organic substances) was developed when laboratory research showed that different organic substances, when exposed to atmospheric conditions, did not react similarly. Specifically, when traces of an individual organic and NO in air were irradiated with artificial sunlight, the resulting levels of smog, in terms of oxidant/ozone yield, eye irritation, plant damage, visibility reduction, etc., were found to vary widely with the chemical structure of the organic reactant. As a result of these studies, the concept of hydrocarbon reactivity has evolved to include several reactivity types, each type corresponding to a specific chemical-biological manifestation of photochemical smog.[6] The reactivity type of interest here is the one associated with the oxidant/ozone yield.

The fact that organic substances differ greatly in reactivity is extremely significant in understanding the photochemical oxidant/ozone-forming process. Such reactivity data are presently available. The data and the methods used for obtaining them are discussed next.

The procedure commonly used for reactivity measurements is to irradiate clean air mixed with the organic vapor, NO, and NO_2 at prescribed concentrations in a smog chamber and to measure the formation of oxidant/ozone. Experimental conditions for such measurements are, to the extent feasible and practical, similar to the conditions typically present in polluted ambient atmospheres. Thus the reactant concentrations, the intensity and spectral characteristics of radiation, the temperature, and often the relative humidity are comparable to actual conditions in the real atmosphere during the summer. Because the concern traditionally has been for the oxidant problems observed in urban areas, nearly all of the reactivity data presently available were obtained under experimental conditions simulating urban atmospheres, and, more specifically, the Los Angeles atmosphere. Recent developments, however, led to recognition of two distinct atmospheric situations (to be referred to here as the urban, or no-transport, situation and the rural, or transport, situation) for which an organic may manifest different reactivities.[27] The terms "urban" or "no-transport" are used to designate the situation in which emissions react for a few hours and cause oxidant problems in the vicinity of their sources. Conversely, in the rural or transport situation, emissions undergo extensive transport and prolonged photochemical reaction and cause oxidant/ozone buildup in distant downwind areas, which may be rural or urban. Again, nearly all of the reactivity data currently available are applicable to the no-transport situation only; there are very few measurements of reactivity under simulated transport conditions.

The scientific evidence detailing the impact that the reactivity of the organic emissions has on air quality within the area of the sources and in the areas far downwind is examined in the following discussion. Scientific evidence pertinent to these questions does exist[25] and is presented next, first for the urban or no-transport situation, and then for the rural or transport case.

Reactivity data obtained through 1965, all applicable to the urban situation, were compiled by Altshuller[3] and presented in terms of a reactivity classification of hydrocarbons and aldehydes, as shown in Table 5-16. Since 1965, several reactivity studies have been reported, including detailed reactivity data on hydrocarbon and nonhydrocarbon organics. These studies and the types of reactivity data reported are listed in Table 5-17.

TABLE 5-16. COMPARISON OF REACTIVITIES OF DIFFERENT TYPES OF ORGANICS[3]

Substances or subclass	Reactivity on 0 to 10 scale						
	Ozone or oxidant	Peroxy-acyl-nitrate	Formal-dehyde	Aerosol	Eye irrita-tion	Plant damage	Overall reactivity
C_1-C_3 paraffins	0	0	0	0	0	0	0
Acetylene	0	0	0	0	0	0	0
Benzene	0	0	0	0	0	0	0
C_{4+} paraffins[a]	0-4	0[b]	0[b]	0	0[b]	0	1
Toluene (and other monoalkylbenzenes)	4	ND[c]	2	2	4	0-3	3
Ethylene	6	0	6	1-2	5	+[b]	4
1-alkenes[d]	6-10	4-6	7-10	4-8	4-8	6-8	7
Diolefins	6-8	0-2	8-10	10	10	0[b]	6
Dialkyl- and trialkyl-benzenes	6-10	5-10	2-4	+[e]	4-8	5-10	6
Internally double-bonded olefins	5-10	8-10	4-6	6-10	4-8	10	8
Aliphatic aldehydes	5-10	+[e]	+[e]	ND[c]	+[e]	+[e]	--

[a] Averaged over straight-chain and branched-chain paraffins.

[b] Very small yields or effects may occur after long irradiations.

[c] No experimental data available.

[d] Includes measurements on propylene through 1-hexene, 3-ethyl-1-butene and 2,4,4-trimethyl-1-1-pentene.

[e] Effect noted experimentally, but data insufficient to quantify.

TABLE 5-17. REACTIVITY DATA

Investigator	Data reported
Altshuller and Bufalini[7]	Reactivity data on hydrocarbons and aldehydes.
Brunelle et al.[15]	Reactivity data for solvent organics.
Dimitriades et al.[31]	Reactivity data for hydrocarbons and aldehydes.
Dimitriades et al.[31]	Rate of NO_2 formation and product yield reactivity data for hydrocarbons.
Dimitriades and Wesson[32] (Bureau of Mines)	Rate of NO_2 formation and product yield reactivity data for aldehydes.
Heuss, J.[36]	Reactivity data on hydrocarbons.
Heuss and Glasson[37] (General Motors)	Eye irritation, rate of NO_2 formation, and product yield reactivity data for hydrocarbons.
Laity et al.[47] (Shell)	Reactivity data for solvent organics.
Levy and Miller[48] (Battelle)	Reactivity data for solvent organics.
McReynolds et al.[56]	Reactivity data for hydrocarbon disappearance.
Miller et al.[57]	Aerosol activity data for hydrocarbons.
Wilson and Doyle[88] (Stanford Research Inst.)	Reactivity data for solvent organics.
Yanagihara et al.[90]	Reactivity data for solvent organics.

Of these studies, however, only a few included systematic testing of a variety of organic compounds. The data from these main studies, summarized in Table 5-18, were used by EPA to classify organics into three reactivity classes, shown in Tables 5-18 and 5-19. The data in Table 5-18 show that organics do differ widely in reactivity.

TABLE 5-18. SUMMARY OF DATA FROM STUDIES ON REACTIVITIES (TOLUENE EQUIVALENTS) AND CLASSIFICATION OF ORGANICS

Organic	Japan EA[a]	BOM[a]	GM[a]	Battelle[a]	Shell[a]	Class
Paraffinic hydrocarbons:						
C_1-C_3 paraffins	0-0.1	--	0-0.2	--	--	I
C_{4+} paraffins	--	--	--	--	--	
Cycloparaffins	0.1-0.7	0.1	0.5-0.6(0.06)[b]	0.4-0.6	0.8-1.0	II
Olefinic hydrocarbons	1.6-2.3	1.6-2.5	0.9-2.0	1.3-1.5	1.8-3.1	III
Aromatic hydrocarbons:						
Benzene	0	0	0.2	0	0.2	I
Primary and secondary mono-alkylbenzenes	0.9	0.8-1.0	0.6-1.0	0.9-1.0	1.0-1.2	III
Tert-monoalkylbenzenes	--	--	0.4	0.6	0.5	II
Dialkylbenzenes	1.4-2.0	1.4-1.5	0.9-1.3	1.0-1.2	1.3-1.7	III
Tri-, tetraalkylbenzenes	--	1.7	1.5	1.5	3.2	III
Styrene	--	--	--	0.7	--	III
Me-styrene[c]	--	--	--	1.5	--	III
Aldehydes:						
Aliphatic aldehydes	--	1.5-2.0	--	--	--	III
Benzaldehyde	--	0.1	--	--	--	I
o-Tolualdehyde	--	0.4	--	--	--	II
m,p-Tolualdehyde	--	0.2	--	--	--	II
Ketones:						
Acetone	0	--	--	0	0.1	I
Me-et-ketone[c]	0.1	--	--	0.6	0.9	I
n-Alkylketones	--	--	--	0.5-0.8	1.4	II
Branched alkylketones	1.4	--	--	1.0-1.8	1.3	III
Cyclic ketones	0.35	--	--	0.2	0.5-0.6	II
Unsaturated ketones	--	--	--	1.5-1.7	--	III
Alcohols:						
Methanol	0	--	--	--	--	I
Ethanol	0	--	--	--	1.0	II
Isopropanol	0.1	--	--	0.2	0.6	I
Primary and secondary $C_{4,5}$ alcohols	0.7-1.0	--	--	--	--	III
Tertiary alkyl alcohols	--	--	--	--	0.3	I
Diacetone alcohol	--	--	--	1.4	--	III
Ethers:						
Diethyl ether	--	--	--	--	2.5	III
Tetrahydrofuran	--	--	--	1.9	1.4	III
Ethyl cellosolves	1.9	--	--	1.5	--	III
Esters:						
Methyl acetate	0	--	--	--	--	
Primary, secondary alkyl C_2+ acetates	0.1	--	--	0.2	0.8-1.0	II
Tertiary alkyl acetates	--	--	--	--	0.5	II
Phenyl acetate	--	--	--	0	--	I
Methyl benzoate	--	--	--	0	--	I
Amines:						
Ethyl amines	--	--	--	0.1-0.2	--	I
N-Me-pyrrolidone	--	--	--	0.7	--	III
N,N-dimethyl-formamide	--	--	--	--	0.2	I
N,N-dimethyl-acetamide	--	--	--	--	0.9	II
Nitroalkanes:						
2-Nitropropane	--	--	--	0.2	0.7	II
Halocarbons:						
Perhalogenated hydocarbons	0.1	--	--	--	--	I
Partially halogenated paraffins	--	--	--	--	--	I
Partially halogenated olefins	0.5	--	--	--	--	III
Halogenated benzenes	0-0.2	--	--	--	--	I

[a] Japan EA = Japan Environment Agency; BOM = Bureau of Mines, U.S. Dept. of Interior; GM = General Motors Corp.; Battelle = Battelle Memorial Institute Laboratories-Columbus; Shell = Shell Oil Co.

[b] Values were obtained from two different GM studies in which test conditions were different.

[c] Me = methyl; et = ethyl.

TABLE 5-19. CLASSIFICATION OF ORGANICS WITH RESPECT TO THEIR OXIDANT-RELATED REACTIVITY IN URBAN ATMOSPHERES

Class I (low reactivity)	Class II (moderate reactivity)	Class III (high reactivity)
C_1-C_3 paraffins[a]	Tert-monoalkyl benzenes	Prim., sec. monoalkyl benzenes
Acetylene[a]	Cyclic ketones	Dialkyl benzenes
Benzene	Tolualdehydes	Styrene
Benzaldehyde[a]	Tert-alkyl acetates[a]	N-Methyl pyrrolidone
Acetone[a]	2-Nitropropane[a]	Partially halogenated olefins
Methanol	C_{4+} paraffins, cycloparaffins[a]	Aliphatic olefins
Isopropanol	Ethanol	Tri-, tetra-alkyl benzene
Tert-alkyl alcohols[a]	Prim., sec. C_{2+} alkyl	Methyl styrene
Methyl acetate[a]	N, N-dimethyl acetamide[a]	Branched alkyl ketones
Methyl benzoate	n-alkyl C_{5+}-ketones[a]	Unsaturated ketones
Ethyl amines[a]		Aliphatic aldehydes[a]
N, N-dimethyl formamide[a]		Diacetone alcohol
Perhalogenated hydrocarbons		Ethers[a]
Partially halogenated paraffins		2-Ethoxy-ethanol
Mono, dichlorobenzenes		
Methyl-ethyl-ketone		

[a] Currently classified as not photochemically reactive under Los Angeles County Rule 66 and similiar regulations.

For a practical application of these differing reactivities, it is necessary that the photochemical behavior of an organic pollutant mixture be consistent with the behavior of the individual components. This requirement was explored in smog chamber studies, and overall results, although not always conclusive, were positive. For example, for an ethane/butane/propylene/NO$_x$ mixture at the organic-to-NO$_x$ ratio of 10:1, removal of the extremely reactive propylene resulted in less smog, which is consistent with the individual component reactivities. At a ratio of 20:1, however, removal of propylene had no effect.[9,44] Smog chamber data on solvent mixtures also showed that less smog was formed when less reactive solvents were substituted for more reactive ones.[29] For the more complex automotive exhaust mixtures, the values for the maximum 1-hr oxidant observed in the smog chamber did not correlate well with the reactivity values computed from exhaust composition and individual component reactivity data, as shown in Figure 5-5.[31] Such lack of correlation, however, was attributed primarily to misidentification of exhaust components and to the inappropriateness of the linear simulation method for calculating mixture reactivities.[31]

Overall, the smog chamber data suggest that the relatively low organic-to-NO$_x$ ratios encountered in typical no-transport atmospheres will probably result in lower reactivity. In the real atmosphere, oxidant formation is expected to be different from that suggested by the chamber data because the main effect of a reduction in reactivity of the organic reactant is a delay in oxidant formation. This delay, in turn, means that the daily oxidant maximum will not be eliminated but, will rather be shifted some distance downwind. Thus the main result to be expected from reduced reactivity is a reduction in the peak oxidant concentration that results from the additional dispersion associated with the time delay. Of course, if the oxidant accumulation is delayed sufficiently, (i.e., until late afternoon), then such accumulation will not occur for lack of radiation. Note, however, that in this latter case, a greater part of the organic precursor will escape the photochemical process during the first solar day and will be transported downwind where, if sufficient NO$_x$ is also present, it will react to form oxidant/ozone. This case is discussed further in a subsequent section of this chapter.

The preceding discussion dealt with the urban or no-transport situation. Analogous evidence on reactivities of organics and on mixture behavior for the rural or transport situation is very scant; therefore, possible answers presented here will have to be surmised for the most part.

Reactivities of organics under simulated transport conditions have not been measured systemically as they have in the case of no-transport. Nevertheless, from the limited direct data available[29,35] and from current knowledge of the oxidant formation mechanism, it has been established that under transport conditions, the effective range of reactivities is more narrow than for the no-transport case. Therefore, and on this

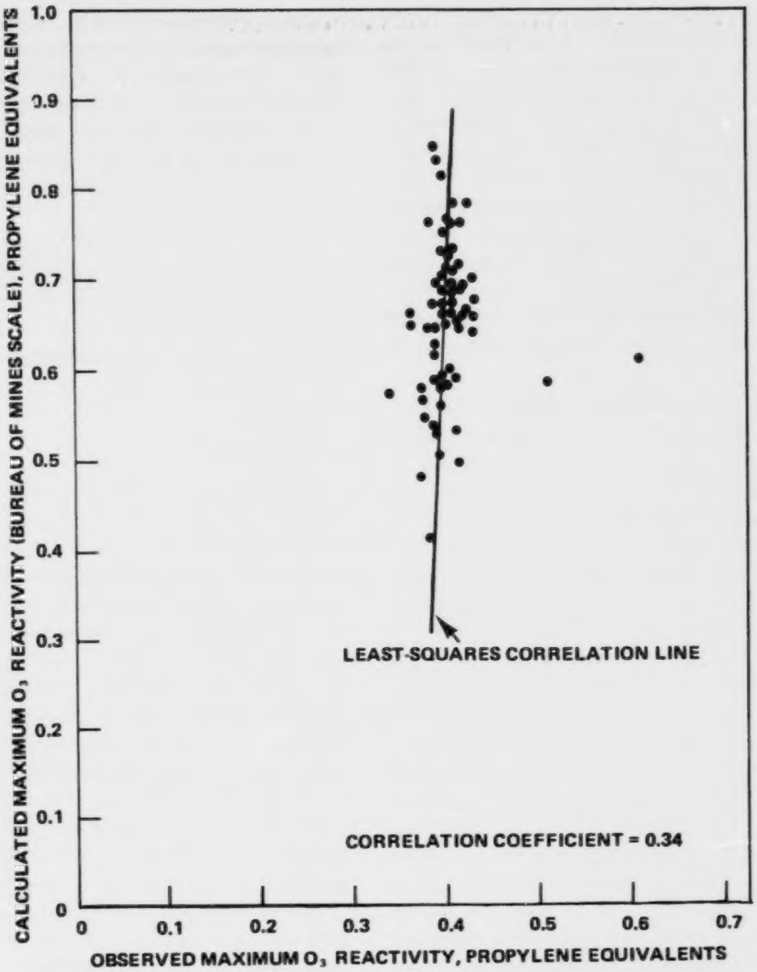

Figure 5-5. Correlation of observed and calculated maximum O_3 reactivities.[31]

basis alone, the impact of emission substitution on air quality well downwind from the sources should be less than the impact within the source area.

The evidence and reasoning discussed in the preceding paragraphs do not preclude the existence of organics that are so slightly reactive that they could be of almost no concern at all insofar as the oxidant problem is concerned. Evidence directly addressed to this possibility is neither adequate nor easy to obtain. One approach to identifying such organics has been offered by EPA recently[29] and is based on the use of smog chambers. Briefly, the method entails smog chamber testing of organics under a variety of reactant concentrations (but within a realistic range), organic-to-NO_x ratio, and prolonged irradiation conditions. Truly nonreactive organics are proposed to be defined as those that, when tested in an appropriate smog chamber do not yield more than 160 $\mu g/m^3$ (0.08 ppm) ozone under any set of test conditions. An alternative method proposed by Chang and Weinstock[20] is based on the use of a photochemical model to predict the role and impact of less reactive organics in transport atmospheres. The two methods were critically examined by Calvert and Jeffries,[17] and their relative merits and limitations were discussed. One problem with the chamber method arises from the presence of chamber wall effects. Such effects create problems; first, because they cause reactivity manifestations that, when extremely unreactive organics are tested, are comparable to or even stronger than those caused by the test reactant mixture; and second, because they are not well understood. Thus, though chamber reactivities of the various organics are probably reliable in a relative sense, their absolute values are somewhat questionable. This latter weakness also affects the chamber-determined reactivity level that separates reactive organics from those of no concern insofar as the oxidant problem is concerned.

The modeling method of Chang and Weinstock[20] also has weaknesses because of the questionable chemical mechanism and kinetic data used by the authors. Furthermore, because such models are derived theoretically, and because their validation is based mainly on comparison with smog chamber data, their predictions cannot be much more valid than those obtained by the smog chamber method. Finally, this modeling method has very limited utility, since photochemical models exist only for those very few organics for which the atmospheric photo-oxidation mechanism has been elucidated.

The key problem here is to identify the boundary organic that separates the reactive organics from the nonreactive ones. Solving this problem would probably require application of both the chamber method and modeling techniques. The chamber method would be used to determine the relative reactivities of organics, and photochemical modeling would be used to predict the absolute reactivity of the boundary organic. Because the chamber data suggest that propane may be a reasonable candidate for the boundary position,[29] and because the mechanism of the atmospheric photo-oxidation of propane is relatively well established, it might be advisable to estimate the oxidant-forming potential of propane using the modeling method.

As an alternative to smog chamber measurement, Pitts et al.[67] suggested that the rate constant for the reaction between the OH-radical and an organic be used as a measure of the reactivity of the organic. Such rate constants have been measured for over 100 hydrocarbon and nonhydrocarbon organics, and with exceptions, their relative values roughly parallel the relative reactivity values obtained by the smog chamber method. Relative to the smog chamber reactivities, these rate constant reactivities are superior in some respects but inferior in others. For example, they are more reliable measures of reactivity in the cases of extremely unreactive organics, and therefore they provide better bases for identifying such organics. On the other hand, this rate constant is not a direct measure of the organic's ability to produce oxidant/ozone; therefore, it has only limited validity.[17]

In summary, the use of the concept of reactivity is generally accepted as sound because organic emissions do differ widely in reactivity. Additional research should be done to provide more reliable bases for identifying those organic emissions that are of no concern insofar as the oxidant problem is concerned.

SUMMARY

Organic pollutants in urban atmospheres consist mainly of hydrocarbons emitted by automobiles and from fuel evaporation, and of oxygenated hydrocarbons from manufacturing and the use of organic chemicals. In urban atmospheres, the ambient total organic concentrations, reported usually as 6- to 9-a.m. averages of total nonmethane hydrocarbons (NMHC), range typically around 1 ppm and can be as high as 10 ppm or even higher (e.g., in Los Angeles). In rural and remote atmospheres, the composition and concentrations of organic pollutants are considerably more uncertain, mainly because of deficiencies in the analytical methods available for the concentrations involved. Overall, the evidence suggests that natural NMHC levels are generally less than 0.1 ppm, a fraction of which consists of vegetation-related terpenes.

Concentrations of NO_x in urban atmospheres vary within a wide range, with highest values exceeding 1 ppm. Such concentrations appear to decline rapidly as the urban air moves away from the city. In rural and remote areas, ambient concentrations do not exceed a few ppb and therefore are often below the 5-ppb detection limit of current commercial NO_x analyzers.

Hydrocarbons and NO_x are emitted into the atmosphere from both natural and manmade sources, with the natural contribution being the greater. This greater contribution, however, does not influence oxidant/ozone formation, because the sources of natural and anthropogenic emissions are spatially segregated, with anthropogenic ones concentrated in the populated areas. At present, mobile sources account for a major part of the organic emissions in most urban centers.

Ongoing controls of mobile source emissions have reduced levels and changed the composition of emissions in favor of the paraffinic component.

Organic emissions differ widely in reactivity (i.e., in their ability to produce photochemical ozone and other oxidants). Thus some organics probably do not contribute to photochemical smog formation. More research is needed to provide reliable bases for identifying those organics that have no bearing on the oxidant/ozone problem.

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6. RELATIONSHIPS BETWEEN AMBIENT OXIDANTS AND PRECURSOR EMISSIONS

INTRODUCTION

This chapter examines the functional relationships observed or theoretically derived between photochemical oxidants (O_x), including ozone (O_3), and the two classes of oxidant precursors, hydrocarbons (HC) and nitrogen oxides (NO_x). These O_x/HC/NO_x relationships are extremely important in that they constitute the basis of the methods for predicting impacts of emissions on air quality.

Since it is now well established that the impact of emissions is not confined solely and entirely within the emission source area, it is necessary to consider emission distributions and related processes that affect the requisite O_x/HC/NO_x relationship. One concept used to clarify this geographic distinction of the emissions effects is the source-receptor relationship, that is, the spatial and temporal relationship between the area and time of emission discharge and the corresponding area and time of oxidant occurrence. Thus the requisite O_x/HC/NO_x relationship(s) must be applicable to the source-receptor relationship encountered in the real atmosphere.

Additional demands on the O_x/HC/NO_x relationships are posed by considerations of utility rather than validity. For example, it would be useful to have valid O_x/HC/NO_x relationships for small-scale applications such as the prediction of localized impact from addition or deletion of an emission source within an urban area.

All of these requirements must be met by the O_x/HC/NO_x relationships if such relationships are to serve their purposes ideally. In reality, however, such ideal validity and utility are unattainable for various reasons. Two of the most important reasons are (1) it is extremely difficult to pinpoint the areas of the specific emission sources that are responsible for the oxidant problem observed in a given locality, and (2) the proposed O_x/HC/NO_x relationships cannot be validated ideally or directly (i.e., by using real atmosphere data without substantial uncertainty). These reasons led to the adoption of the following two premises: first, perfectly accurate relationships will never be obtained; and second, the development of usable O_x/HC/NO_x relationships will have to be pursued in the form of an iterative process in which judgment developed from and during the use of less accurate relationships is fed back into the development of improved ones.

Consistent with these premises, relationships between oxidant/ozone-related air quality and emissions have been pursued following three distinct approaches that differ mainly in degree of empiricism. In order of decreasing empiricism, these approaches are as follows:

1. Empirical Approach. This approach entails statistically or nonstatistically associating ambient oxidant-related air quality data either with ambient concentrations of precursors or with precursor emission rates. These associations are clearly not cause-effect in nature, and their intended use is not to predict absolute air quality; rather, it is to estimate changes in air quality resulting from changes in emission rates.
2. Mechanistic Models of O_x/HC/NO_x. This approach entails deriving cause-effect relationships between oxidant and precursors through laboratory testing and chemical mechanistic simulations. As in the preceding case, this approach is intended to predict only changes in air quality resulting from changes in emission rates.
3. Air Quality Simulation Model (AQSM) Approach. This approach entails deriving the requisite air quality-emission relationships through mathematical representation of the transport, dispersion, transformation, and deposition processes.

Its intended use is to predict absolute levels of air quality from a given emission rate and meteorological data.

The oxidant-emissions relationships or models developed to date through all these approaches are applicable only to the urban oxidant problem, and more specifically, to situations in which the geographical dimension of the source-receptor relationship is comparable to that of the urban area. Models relating local emissions and oxidant concentrations (caused by these emissions) in distant downwind areas are currently under development.

Currently available models have received only limited testing; their validity, therefore, is only limited. Furthermore, assessments made of these models do not reflect a consensus of opinion. The problems in validating air quality models are considered to be prohibitive by some investigators, but not insurmountable by others.[14]

The following sections will cover the subject of the oxidant/ozone-emissions relationships by describing and discussing first the simpler empirical models and then the more sophisticated ones. Consistent with the previously stated premise that such models will never be perfect, the emphasis in the following discussions will be placed on the relative merits and drawbacks of the various models rather than on their absolute validity and utility. For a more comprehensive coverage of models for predicting air quality, the reader is referred to a recent report by the National Academy of Sciences.[38]

It should be noted that of the O_x/HC/NO_x relationships currently derived and reported, some pertain to oxidant, as measured by the potassium iodide method (see Chapter 7), and some pertain to ozone. Relationships derived from the chemical mechanism of the atmospheric photochemical process pertain to ozone.

MODELS BASED ON EMPIRICAL RELATIONSHIPS

Rollback Model

To relate precursor emission rates to oxidant or ozone air quality, it is necessary that two main processes be quantitated: (a) the combined process of dispersion and sink-removal of precursor emissions, and (b) the combined process of photochemical formation and sink-removal of oxidant/ozone. One proposed method for quantitating the dispersion-removal process is by assuming a simple linear rollback model expressed by the following equation:[7]

$$c_i = b + ke \qquad (6\text{-}1)$$

where c_i is the concentration of a pollutant at point i in the ambient air, b is the background concentration of the pollutant, e is the pollutant emission rate, and k is a constant dependent on meteorology, location of sources relative to point i, and on other factors (e.g., sinks) affecting the impact of the sources at point i. Equation (6-1), after processing and simplification,[7] yields equation (6-2):

$$R = \frac{(gf)\,(PAQ) - Std}{(gf)\,(PAQ) - b} \times 100 \qquad (6\text{-}2)$$

where R is the percentage reduction needed to achieve the standard; PAQ and Std denote present and desired air quality (standard), respectively, in terms of pollutant concentration; and gf is the emission growth factor.[7] In the case of oxidant or ozone, the values for PAQ and Std are the second highest oxidant or ozone concentration observed in the base year and 160 $\mu g/m^3$ (0.08 ppm), respectively. By inserting those values and values for gf and b, calculation can be made of the percent reduction of oxidant/ozone needed to achieve the air quality standard of 160 $\mu g/m^3$ (0.08 ppm) of ozone. If ambient oxidant/ozone concentration is assumed to be proportional to the reactive organic emission rate, then the calculated oxidant/ozone control requirement equals numerically the control requirement for reactive organic emissions. Reactive organics are usually meant to include all hydrocarbons except methane; however, other reactivity definitions have been used in which other organics as well as methane were exempted as being nonreactive.[60]

The preceding method for relating emissions to air quality is known as the simple or linear rollback model. It has several limitations, the main ones arising from the the assumptions used in the quantification of both the dispersion process and the photochemical process.[7] Thus the dispersion-related equations (6-1) and (6-2) cannot be validated experimentally, since such validation would require that emission rates for each and every source be changed (reduced) identically, a requirement that obviously cannot be met in real situations. Also, the assumption of proportionality between oxidant and reactive organic emission rates is of questionable validity, as it is not supported by either smog chamber[11] or theoretical evidence.[37,47] Finally, the method ignores the role of the NO_x precursors, which have been established as exerting an important role. Despite these and other limitations, the simple rollback method has received attention[56,57] mainly because of its computational simplicity and its relatively small demands for input information. In terms of accuracy and validity, this model is clearly the crudest of those currently available.

Modified Rollback: Observational Model

The main difference between simple and modified rollback models is in the quantification of the photochemical process. Thus, although in the simple rollback the percent control needed for HC emissions is taken to be equal to the percent control needed for oxidant/ozone, the modified rollback model utilizes an oxidant-to-precursor dependency derived from aerometric data. This latter derivation and resulting oxidant-to-hydrocarbon dependency is often referred to as the observational model and is the quantitative basis of EPA's first proposed method, the Appendix-J method, for calculating oxidant-related control requirements.[59] The observational model has been described and discussed in detail elsewhere;[1,50] therefore, only a brief description will be included here.

The observational model is based on the assumption that early morning precursor concentrations are indicators of the oxidant levels that will occur later in the day. Consistent with this assumption, aerometric data taken in several urban centers were used to draw the upper limit curve shown in Figure 6-l. Specifically, this curve was constructed by plotting daily maximum l-hr oxidant concentrations against 6- to 9-a.m. average nonmethane hydrocarbon (NMHC) concentrations. The curve was drawn through the uppermost points. Note that both the oxidant and NMHC data were taken at the same monitoring site—Continuous Air Monitoring Program (CAMP) site—invariably located in the downtown area of the respective urban center. Thus the upper limit curve is taken to depict the relationship between the hydrocarbon precursor and the oxidant formed within source-intensive areas under those meteorological conditions that are most conducive to oxidant formation (i.e., clear skies, high temperature, etc.).

Accepting the upper limit curve as depicting the quantification of the photochemical process, control requirements for oxidant reduction can then be calculated using the rollback equation (6-2) as follows: From the curve in Figure 6-1, one can read off the NMHC value corresponding to PAQ (that is, to the second highest oxidant concentration observed in a location) as well as the NMHC corresponding to the standard (that is, to 160 $\mu g/m^3$, or 0.08 ppm). This latter NMHC was determined to be 0.24 ppm C, and it is the air quality standard for hydrocarbon (to be used as a guide only and not as a true air quality standard).[58] By using these NMHC values and assuming b = o (i.e., no background oxidant or NMHC),[58] the percentage control required for NMHC emissions was calculated as a function of oxidant concentration, and the resultant function was published by EPA in the form of the Appendix-J curve.[59] A somewhat different approach was used by Schuck and Papetti to construct an upper limit curve specific for Los Angeles.[60] By this latter approach, the NMHC values used were averages of data taken at several sites in the Los Angeles Basin; whereas, the oxidant values used were those for the highest oxidant concentration observed anywhere in the Basin. This Los Angeles curve is shown in Figure 6-2.

This modified rollback, or Appendix-J, model has been critiqued extensively.[9,13,24] Briefly, the model has limitations and advantages related (1) to the rollback equation (6-2), (2) to the assumption of zero background oxidant or NMHC, and (3) to the upper limit curve. Limitations and justification of the rollback equation are discussed in the preceding section. The assumption that there is no background oxidant or NMHC is clearly incorrect, but it has been adopted in the interest of simplicity and because reliable data on the level of such background are lacking. The limitations and advantages of the upper limit curve are summarized as follows:

1. The curve probably depicts the dependence of oxidant on the dispersion factor rather than the dependence on the hydrocarbon reactant factor.[13]
2. The curve depicts a purely empirical relation, not a cause-effect one, and it, therefore, cannot automatically be assumed to have predictive value; (it would have a more cause-effect nature if oxidant were measured within the same air mass in which the HC and NO_x measurements were made).
3. The curve disregards the NO_x factor.
4. Experimental error makes the low end of the curve (air quality standard for NMHC) highly uncertain.

5. Scarcity of data points makes the upper part of the curve highly uncertain.
6. The curve is not necessarily valid in locations other than those from which it was derived.
7. The curve disregards the oxidant transport phenomena.
8. The curve has no statistical nature.

Justification of the upper limit curve is mainly based on the following:

1. The curve is derived from real atmospheric data, which is a more realistic alternative to laboratory smog chamber data, for example.
2. The curve is in qualitative agreement with the smog chamber data, at least insofar as the oxidant-to-hydrocarbon dependence is concerned (Figure 6-3).
3. The curve can be improved with acquisition of additional data.

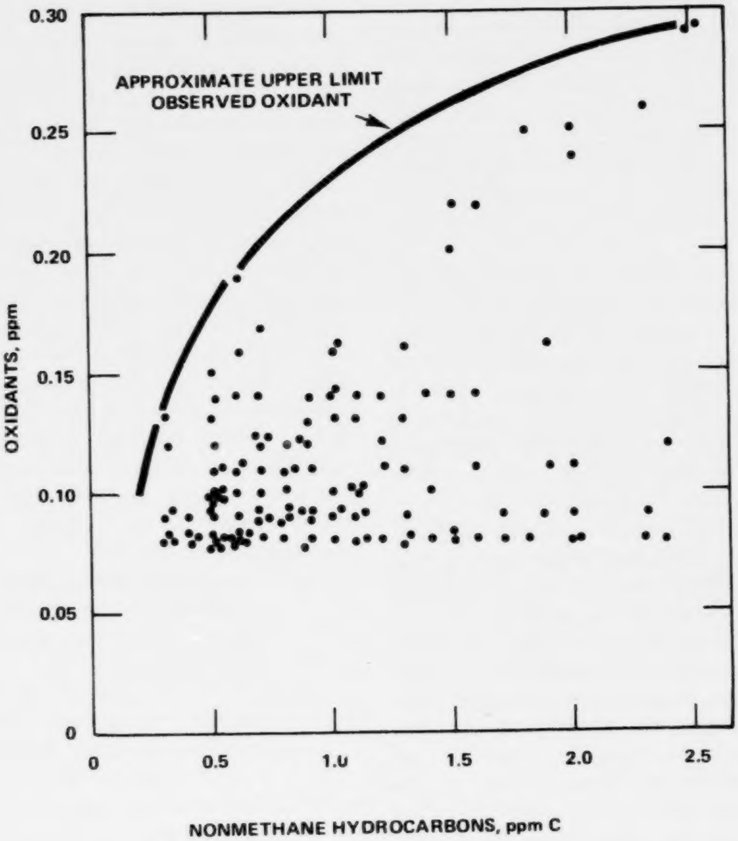

Figure 6-1. Maximum daily 1-hr average oxidants as a function of 6-to-9-a.m. average of nonmethane hydrocarbon (CAMP data from four U.S. cities).[150]

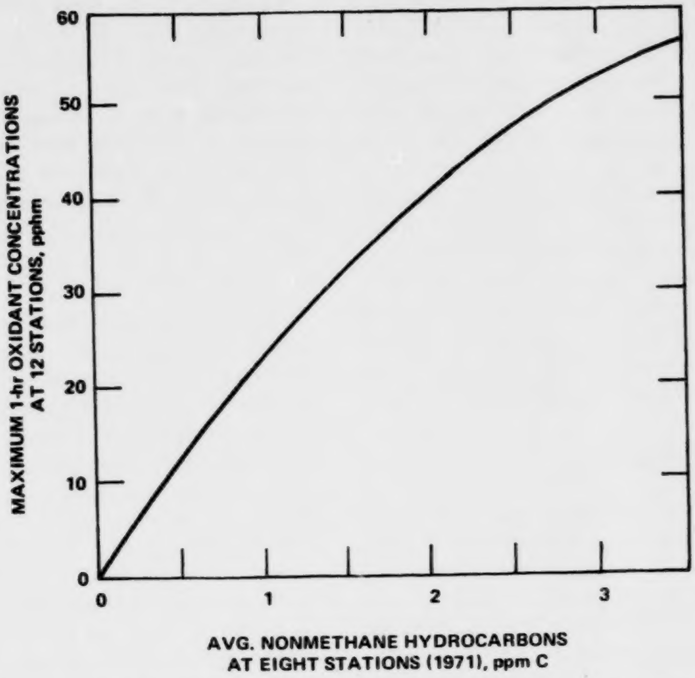

Figure 6-2. Upper limit oxidant values in the Los Angeles south coast air basin as a function of average 6-to-9-a.m. hydrocarbon concentrations.[60]

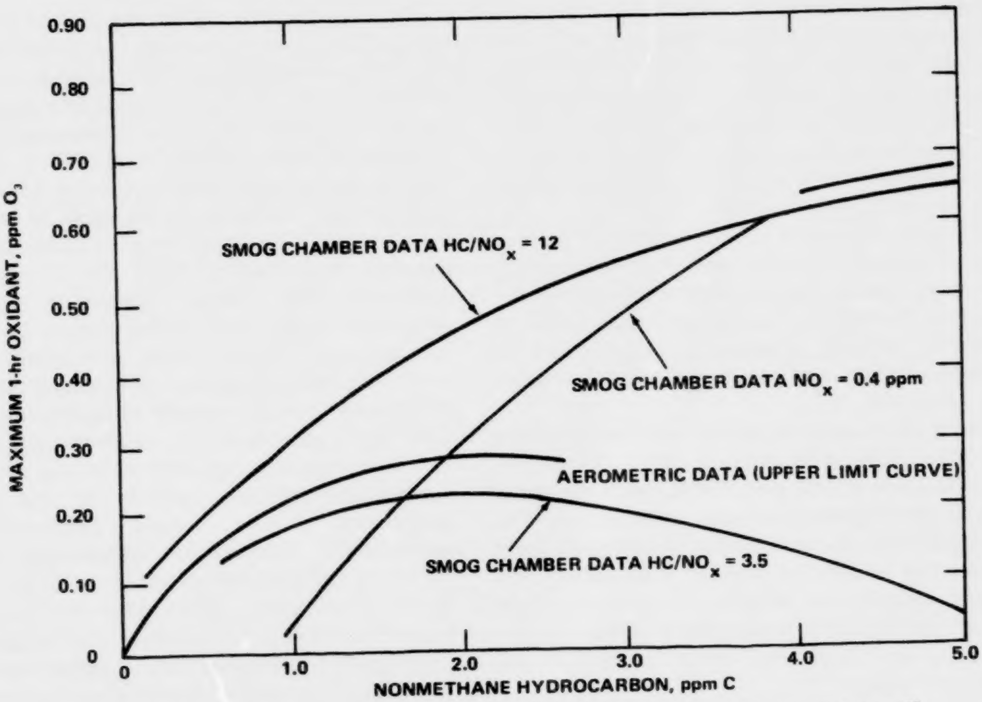

Figure 6-3. Oxidant-hydrocarbon relationships from smog chamber and aerometric data.[13]

Overall, the Appendix-J method is judged to be no more valid than the simple rollback model because it is based on relatively few data taken from several cities. The method has more validity when it is derived from and used in one and the same locality, as, for example, in the case of the Schuck-Papetti curve for Los Angeles. This, however, requires an abundance of data, provided by multiyear rather than multistation measurements. Such data are currently available only for a very few cities.[1] Lack of adequate data, ambient measurement error, and influences associated with siting of the monitoring stations are probably the main reasons for the rather large differences observed among individual location upper limit curves.[61]

Statistical-Empirical Models

Statistical-empirical models are based on statistically defined associations among ambient air quality indices, meteorological parameters, and emissions rates. A considerable amount of work has been done in establishing such associations. However, much of that work was addressed to the air-quality-to-meteorology dependency and the development of predictive models for short-term episode forecasting. Such models are not relevant to the subject of this document. Relevant models are those addressed to the air-quality-to-emission dependency—that is, models intended to predict long-term impact of emission controls on oxidant air quality and to calculate control requirements for oxidant reduction. The most notable of these models were recently reviewed by Myrabo et al.[35] The following discussion of such models is based partly on the Myrabo review.

With one exception, the statistical-empirical models relating ambient oxidant to precursors neglect the spatial and temporal distribution of the precursor emissions. Accordingly, their utility is limited to relating changes in total levels of emissions within a region to changes in region-wide air quality.

Merz et al.[33] used regression analysis to relate daily 1-hr maximum oxidant to 6- to 9-a.m. concentrations of NO_x and NMHC, with the latter taken to be 50 percent of total hydrocarbon. The data used were for downtown Los Angeles, and only for the months of August, September, and October. Results are shown in Figure 6-4. The diagrams of Figure 6-4 indicate that NO_x control would have a slight but beneficial impact on oxidant air quality.

Kinosian and Paskind[26] also used July-September data from Los Angeles to relate, through regression analysis, daily maximum 1-hr oxidants to 6- to 9-a.m. NO_x for constant NMHC. The NMHC data, again, were not obtained directly; they were computed from total hydrocarbon (HC) data using established HC-to-NMHC relationships. Results from four Los Angeles sites are shown in Figures 6-5 through 6-8. These results, unlike the Merz et al. model,[33] indicate that NO_x control, unless drastic, is detrimental to oxidant air quality.

Finally, Trijonis[55] used a stochastic model to relate emission rates in downtown Los Angeles (DOLA) to 7:30-9:30-a.m. ambient concentrations of hydrocarbon and NO_x, which were then related to the oxidant concentrations observed in the sections of the Los Angeles Basin lying downwind from DOLA. Specifically, Trijonis used average values of oxidant concentrations measured at DOLA, Pasadena, and Burbank, weighted according to direction and speed of the morning-to-noon wind. Trijonis expressed his results in terms of annual exceedance of the California oxidant standard (0.1 ppm as ozone) as a function of NO_x and reactive hydrocarbon emission levels in DOLA (Figure 6-9).[55] These results again show NO_x control to have a detrimental impact on oxidant air quality, at least for Los Angeles.

All three models discussed in the preceding paragraphs are not capable of treating spatial resolution of emissions. Thus, by those models, a region or urban center is treated as a single point source, a simplification that is obviously helpful, but that is also penalizing in that it makes it impossible to deal with questions of localized impact from addition or deletion of emission sources within the urban area. To lessen this latter penalty, investigators from the University of California, San Diego, offered a statistical-empirical model with some spatial resolution incorporated.[5,34] Specifically, their model related oxidant air quality (expressed in terms of number of hourly violations of the Federal oxidant standard of 0.08 ppm as ozone) to reactive hydrocarbon emission levels integrated over the prevailing windstream corridor leading to the oxidant monitoring station. Spatial resolution was achieved by defining the windstream corridor width to be approximately one-tenth of the maximum dimension of the urban area. Using data from 17 stations in San Diego and Los Angeles, the investigators generated a hydrocarbon-emission-to-air-quality relationship that they then proposed

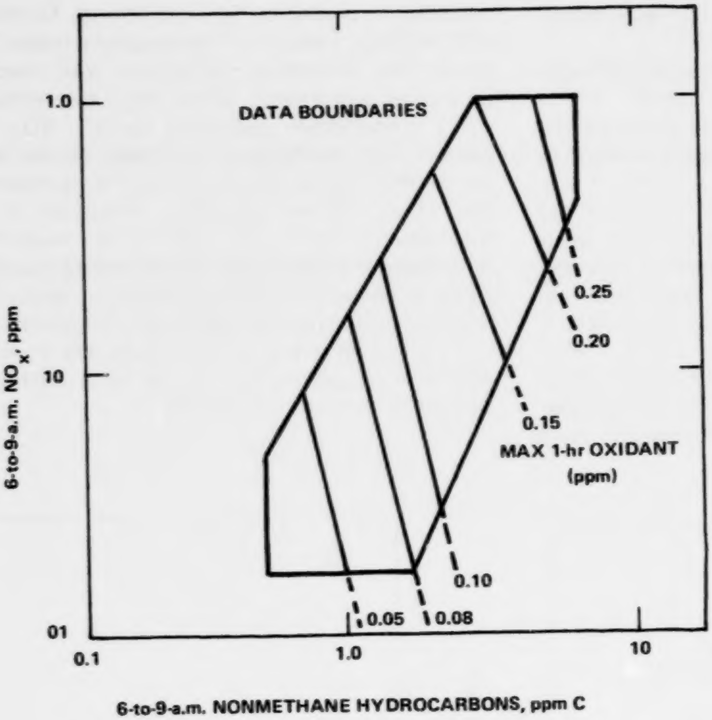

Figure 6-4. Merz, Painter, and Ryason's relation of NO_x and NMHC assumed as 50 percent of total HC and oxidant for downtown Los Angeles.[33]

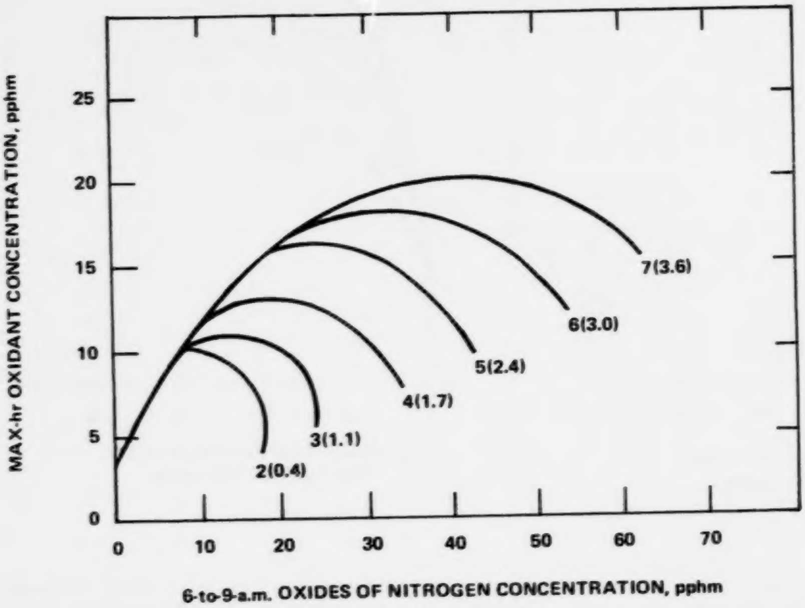

Figure 6-5. California Air Resources Board aerometric results, relation between 6-to-9-a.m. NO_x, 6-to-9-a.m. HC, and maximum hourly oxidant concentrations in downtown Los Angeles. (Individual curves show total and NMHC concentrations in ppm.)[26]

for use for predictive purposes. The NO_x factor, obviously, is ignored by this model.

All the statistical-empirical models described in the preceding paragraphs, and other similar ones,[2,38] are relatively simple in that they treat the conversion of emissions into ambient oxidant as one process. Thus distinct components of this process such as the vertical dispersion of emissions within a definable atmospheric layer (mixing layer), local (intracity) transport by wind, and chemical reactions are not delineated and treated separately. Because of this simplification, these models lose much of their validity when applied to areas with characteristics widely different from those of the area for which they were developed (for example, mixing heights, wind patterns, or photochemical conditions). To reduce this problem, Tiao et al.[54] developed a model that treats the dispersion, advection, and reaction processes separately, using separate terms in mass conservation equations for NO, NO_2, and ozone. The coefficients of these terms were determined statistically by fitting the equations to the observations available. Because of its mechanistic detail, the Tiao et al. model[54] is inherently more valid than the preceding models in treating diverse situations. However, because it does not include spatial resolution, this model, like the preceding ones, is not applicable in areas where a major fraction of the local oxidant is caused by extraneous sources.

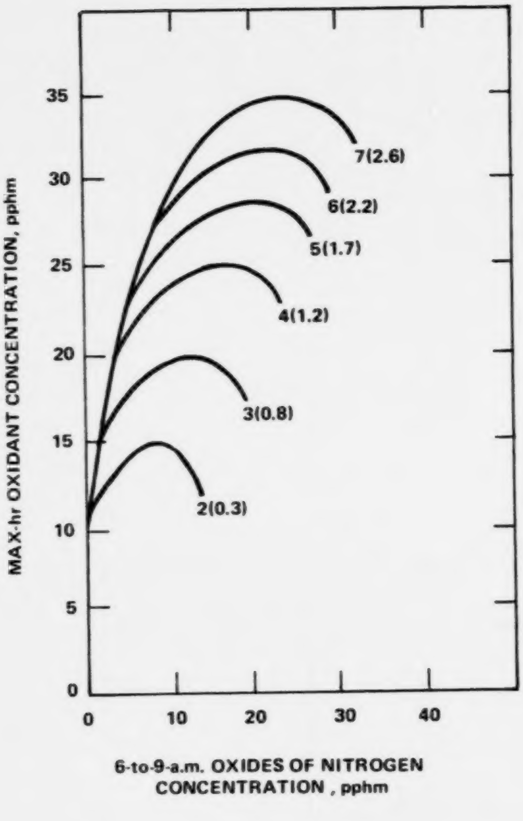

Figure 6-6. California Air Resources Board aerometric results, relation between 6-to-9-a.m. NO_x, 6-to-9-a.m. HC, and maximum hourly oxidant concentrations in Azusa. (Individual curves show total and NMHC concentrations in ppm.)[26]

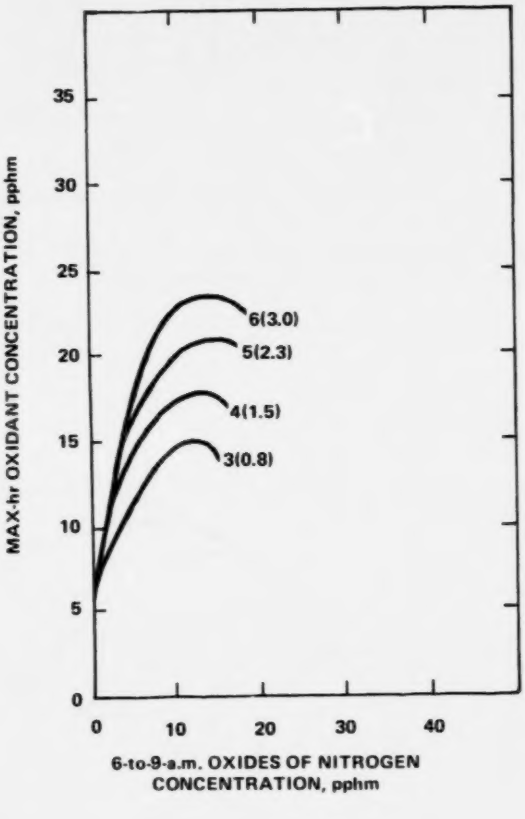

Figure 6-7. California Air Resources Board aerometric results, relation between 6-to-9-a.m. NO_x, 6-to-9-a.m. HC, and maximum hourly oxidant concentrations in San Bernardino. (Individual curves show total and NMHC concentration in ppm.)[26]

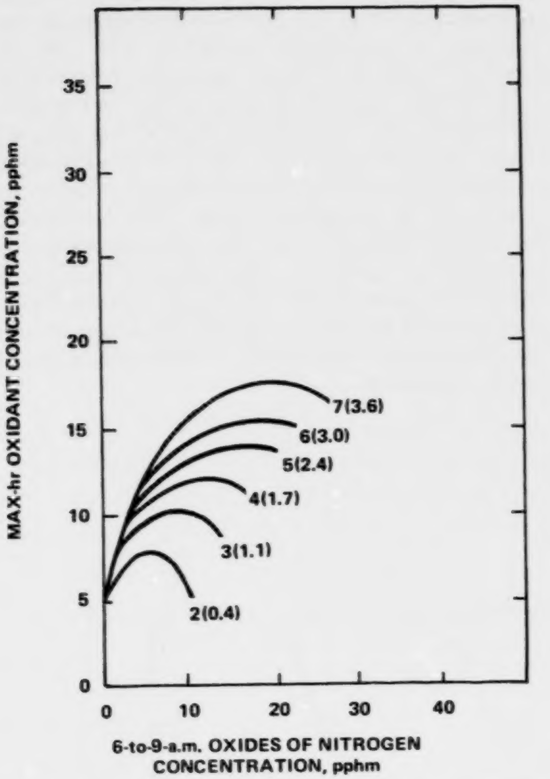

Figure 6-8. California Air Resources Board aerometric results, relation between 6-to-9-a.m. NO_x, 6-to-9-a.m. HC, and maximum hourly oxidant concentrations in Anaheim. (Individual curves show total and NMHC concentrations in ppm.)[26]

The statistical-empirical models have advantages arising from the fact that the models are constructed from real atmospheric data and are statistical in nature. Furthermore, such models are computationally simple and inexpensive, and unlike the rollback models, most of them consider both precursor factors, HC and NO_x.

Limitations of the statistical-empirical models arise mainly from the stochastic nature of such models. Because they are based on associative rather than cause-effect relationships, such models have limited predictive validity, especially when predictions call for extrapolating the model beyond the range of data from which it was derived. Furthermore, the models that relate oxidant to ambient concentrations rather than to the emissions of precursors are subject to the same validity limitation as the Appendix-J method, namely, that the model does not directly relate air quality to precursors. Instead, the direct relationship is between oxidant and the atmospheric dispersion factor. In general, the statistical-empirical models have less validity when they are derived from and are applied to a variety of areas with significantly different meteorology and emission characteristics.

In conclusion, when all the strengths and limitations of the statistical-empirical models are evaluated, such models are judged to be superior to the rollback models, especially when they are designed for local use.

MECHANISTIC MODELS OF O_x/HC/NO_x RELATIONSHIPS

The development of reaction mechanisms describing the photo-oxidation processes of hydrocarbons and oxides of nitrogen has evolved extensively through the use of experimental data from smog chamber studies.[10] A basic understanding of the O_x/HC/NO_x chemical relationship is a prerequisite to developing techniques for relating emission to ambient oxidant levels. It is important, therefore, that smog chamber studies be performed under conditions as similar as possible to those of real polluted atmospheres. Thus, considerations such as light intensity and its spectral distribution, temperature, humidity, and composition and concentration of reactants should be comparable to those in typical polluted atmospheres. The utility of one such smog chamber data set exhibiting many of the abovementioned attributes has been demonstrated via the development of a chemical kinetic model for predicting impacts of emission change on air quality and for calculating ozone-related control requirements.[8] This technique, given the acronym EKMA for Empirical Kinetic Modeling Approach, is one of several discussed by EPA in their report, *Uses, Limitations and Technical Basis of Procedures for Quantifying Relationships Between Photochemical Oxidants and Precursors.*[39] Since application of the technique for air quality control is covered in detail in this work, only the scientific basis of the technique will be considered here.

EKMA is based on the ozone-to-precursor relationship (O_3/HC/NO_x) derived from a smog chamber study of automotive exhaust mixtures.[11] The selection of this study over several other smog chamber data sets in existence [11,40] was primarily based on the rather representative nature of the experimental conditions to that of urban polluted atmospheres.[10] Although the quality of the selected data set is considered high, deficiencies nevertheless still exist, as follows:

1. The selected data are limited to those for hydrocarbon-to-NO_x reactant ratios ranging from 1:1 to 12:1, a range that does not extend to sufficiently high values.
2. The ozone yield data are erroneously high for reactant concentrations close to zero, because at such low concentrations, the chamber background reactivity becomes important relative to the reactivity of the test mixture.
3. The selected data were obtained under fixed radiation intensity conditions in contrast to the diurnally varying sunlight intensity in the real atmosphere.

Consideration of these and other minor deficiencies as well as practical constraints on the number of parameter variations that can be studied in smog chambers lead to the utilization of a photochemical kinetic mechanism to simulate and project the experimental data. The detailed photochemical mechanism was first fit to the smog chamber data on automotive exhaust mixtures and then used to extrapolate ozone yields for varying initial hydrocarbon and NO_x concentration and irradiation conditions. Resulting $O_3/HC/NO_x$ relationships for conditions similar to those in the Los Angeles atmosphere during the smog season are depicted in Figure 6-10 in terms of a family of ozone isopleths.

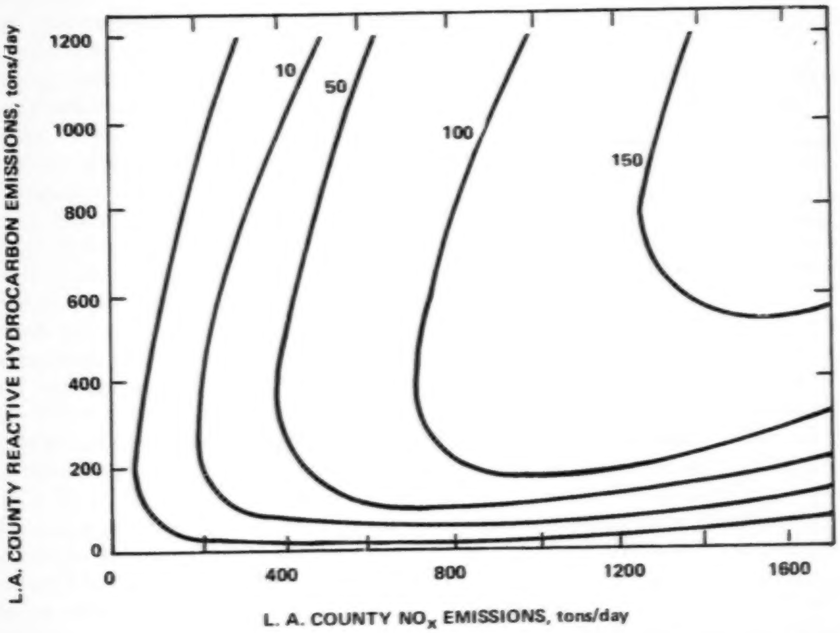

Figure 6-9. Expected number of days per year exceeding 0.10 ppm versus NO_x and reactive hydrocarbon emission levels for central Los Angeles.[55]

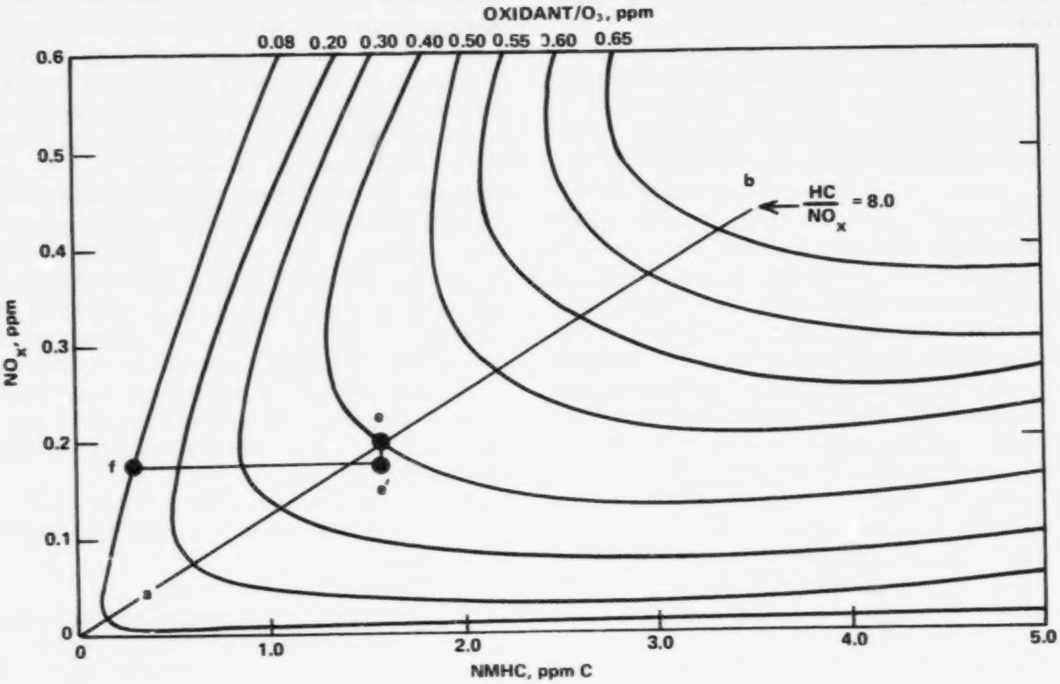

Figure 6-10. Oxidant/O_3 isopleths derived from combined use of smog chamber and photochemical modeling techniques.[16]

The chemical kinetic modeling technique[16] used to derive the isopleths of Figure 6-10 is based on a 75-reaction-step mechanism for a propylene and n-butane hydrocarbon mixture. The two-component hydrocarbon system is a surrogate for the automotive exhaust hydrocarbons, which in turn is a surrogate for atmospheric organic pollutant mixtures. Seventeen smog chamber experiments were modeled in which the agreement between observed and model-predicted ozone yields was maximized by optimizing the butane-to-propylene ratio in the model. Chamber background reactivity was simulated in the model by assuming a source of propylene resulting from desorption from the chamber walls. After optimization, the model was extended to consider conditions more realistic to urban polluted atmospheres. Major changes in the photochemical kinetic model included removal of the chamber background reactivity steps, consideration of diurnally varying photolytic reaction rate constants, simulations over 9-hr irradiation periods corresponding to the hours between 7 a.m. and 4 p.m., and consideration of dilution rate more appropriate to that of an urban atmosphere.

With these adjustments, the model-generated isopleths shown in Figure 6-10 are taken to represent roughly the atmospheric situation in the Los Angeles Basin. The approximate nature of the representation arises since the isopleths, despite the model adjustment, reflect biases caused by the simplifications and possible perturbations inherent to the smog chamber experiment. Thus, for example, the absence of continuous fresh emission influx in the smog chamber simulation is one simplification that may have introduced a bias. The isopleths may also reflect biases introduced by unknown effects of the chamber walls, by use of unrealistic dilution and reactant mixing conditions in the smog chamber test, and possibly by other factors as well.

In light of these uncertainties, the ozone isopleths derived from this model (Figure 6-10) are not assumed to have absolute validity and therefore should not be used to predict ambient ozone levels from given ambient 6- to 9-a.m. concentrations of NMHC and NO_x or vice versa. Application of the isopleths is considered, in a relative sense, as illustrated in the following example calculation for emission control requirements. The detailed reasoning behind this calculation procedure and its advantages are given elsewhere.[11]

Control requirements for achievement of the ozone and NO_2 standards are calculated for a region from the second highest 1-hr oxidant/ozone concentration observed in the region's atmosphere, from the NMHC-to-NO_x ratio during 6 to 9 a.m., and from the annual NO_2 mean concentration. For example, if the ozone concentration, the NMHC-to-NO_x ratio, and the annual NO_2 mean values are 0.4 ppm, 8.0 ± 4.0, and 0.06 ppm, respectively, then the isopleth counterparts of the ambient NMHC and NO_x concentrations causing the 0.4 ppm of ozone are, on the average, those defined by the point e; that is, the intercept of the 0.4 ppm ozone isopleth with the constant slope line ab corresponding to a NMHC-to-NO_x ratio equal to 8.0 (Figure 6-10). Next, the NO_x reduction needed for achieving the national ambient air quality standard (NAAQS) for NO_2 (annual mean of 0.055 ppm) is calculated to be 10 percent, assuming proportionality between ambient NO_x reduction and ambient NO_2 reduction. Reducing NO_x by 10 percent defines point é. For achievement of the NAAQS for ozone, the NMHC must then be reduced along the line éf to the point of being the intercept of line éf with the 0.08 ppm ozone isopleth. Such a NMHC reduction is calculated to be 81 percent. Similar calculations for the upper and lower limits of the NMHC-to-NO_x ratio range give NMHC control requirements equal to 62 percent and 87 percent, the latter control being the one that will achieve the ozone standard regardless of NMHC-to-NO_x ratio.

Note that the EKMA is applicable only in those situations in which the source-receptor relationship is known and temporally consistent with that implied in the model. Thus since the model predictions are for 9-hr irradiation of emissions and the computed ozone peak appears usually after 5 to 6 hours of irradiation, the model-predicted control requirements are only for emission sources lying within an upwind-distance equivalent to 5 to 6 hr (at the most, 9 hr) of wind transport. Situations in which a major fraction of the observed ozone appears to be caused by emissions from more distant upwind sources or from transported ozone aloft (anthropogenic or stratospheric in origin) cannot be appropriately treated by this model.

The possibility of separating out the ozone contribution of the extraneous sources and of defining the local ozone component on which the isopleths are applicable has been explored, but without much success. At this time, the best judgment that can be made on this question is only qualitative. One of many conceivable scenarios describing the interaction between extraneous ozone and local ozone formation is as follows. Local emissions are discharged in the early morning into a relatively thin, stable layer (100 to 200 m) of ozone-free air. As the day progresses, this inversion is eroded, and the reacting emissions disperse into the subsidence inversion layer that contains the extraneous ozone. By early afternoon, vertical mixing is complete, and pollutants (local and extraneous) are confined in a single layer under the subsidence inversion. For such a scenario, the extraneous ozone can be assumed to have undergone the following reduction processes:

1. Dilution resulting from mixing with the ozone-free air in the radiation inversion layer.
2. Destruction on surfaces.
3. Destruction in reaction with precursors and other reaction mixture constituents.

In contrast, this same extraneous ozone also enhances local oxidant formation processes. The quantification of the net impact of the extraneous ozone on ground-level ozone concentrations is beyond the capabilities of the EKMA approach and lies in the realm of the air quality simulation model. Nevertheless, there are some indications that the destructive processes outweigh those that enhance ozone formation. Thus, for example, Seinfeld performed modeling tests[51] that showed that for an urban scenario similar to the one described here, extraneous ozone had a positive effect on ground-level ozone concentration, but that such an effect was less than additive. Therefore, subtracting the entire extraneous ozone concentration (measured upwind or aloft) from the maximum ozone concentration observed in an urban center may introduce substantial error in estimating the local ozone component for which the isopleth method is applicable. Conservative first-order approximation estimates can be made in areas subject to extraneous ozone by assuming negligible impact from the extraneous source. Further guidance regarding such calculations is provided elsewhere.[41]

From the preceding discussion, it is clear that the EKMA is not ideally applicable to situations in which the oxidant problem is largely of extraneous origin. The question that needs to be examined next is whether, with the exception of these situations, the same set of isopleths can be used everywhere within the continental United States. Possible reasons for concern are that the meteorology- and emission-related factors that affect local ozone formation (and hence the ozone-to-precursor relationships) differ from location to location. Thus prevailing sunlight intensity, temperature, and dispersion conditions in Los Angeles are different from those in Chicago, for example. Likewise, the organic emission composition, organic-to-NO_x ratio, and emission discharge patterns may be different for various urban areas. To explore the sensitivity of the EKMA to different urban areas with widely diverse conditions, the method was subjected to a sensitivity test,[17] as follows. First, new sets of isopleths were generated by changing the model parameters related to light intensity, dilution rate, hydrocarbon composition (i.e., propylene-to-butane ratio), and emission discharge after 9 a.m. These new sets of isopleths were then used to compute emission control requirements for given ozone reduction strategies. Results showed that although the various sets of isopleths differed in isopleth shape and spacing, the control requirement estimates were nearly identical for all of them. Thus the same set of isopleths could be used in different locations without introducing a substantial error in the calculation of control requirements. However, given that such error was substantial, it could be reduced by adjusting the appropriate model parameters and generating a new set of isopleths that would more closely reflect the prevailing conditions in the region.

Note that the sensitivity to parameter variations is that of the model and may not necessarily reflect responses of the real atmosphere. Therefore, this technique, like all others, must be verified as to its predictive accuracy.

To complete the discussion on the modeling $O_3/HC/NO_x$ relationships, it should be added that a model has been proposed by Los Angeles County investigators[12] that combines smog chamber and aerometric data. The method has been discussed in detail by Souten et al.,[52] who have pointed out several weaknesses in the approach. Because of these weaknesses, the model has not received much attention.

In concluding this discussion on the mechanistic $O_3/HC/NO_x$ models, it is stressed that such models, although clearly superior to the rollback and statistical models, are also acknowledged to have several imperfections, as discussed elsewhere.[8,11,18] Briefly, the specific model described here, (EKMA), has a conceptual limitation arising from the fact that the ambient atmosphere, with all its complexity, cannot be duplicated in the smog chamber. Furthermore, the model does not predict frequency of occurrence of ozone concentrations. The use of photochemical modeling techniques in deriving the EKMA $O_3/HC/NO_x$ relationships (Figure 6-10) has increased the utility of these relationships, but the photochemical model used also has its imperfections. The EKMA model is also limited in that it requires input information, some of which is not available. Thus the required NMHC-to-NO_x ratio data are not commonly available, mainly because measurement of ambient NMHC is not required by law. Finally, the model does not have the spatial and temporal resolution needed to treat distribution of ozone within an urban area.

All of these imperfections are real and of consequence, but certainly they do not altogether prohibit the use of this model. Viewing these imperfections in the context of the premise that a model shall be judged mainly in relation to other existing models, it would appear that the model described here is somewhat superior to the others presently ready for use. Thus many of the model's imperfections are also shared by the rollback and empirical-statistical models, whereas some of its strengths are not. For example, the strongest points of this model are the cause-effect nature of the model's ozone-to-precursor relationships, and the quantitative consideration of the NO_x-precursor factor. The only models that have both these features are the air quality simulation models. Imperfections of the photochemical model used to supplement and adjust the smog chamber data are not critical since the photochemical model is validated against laboratory data. Thus, future changes in the chemical mechanism of the photochemical model can have only a small effect on the isopleths and an even smaller effect on control requirement estimates computed from the isopleths.

AIR QUALITY SIMULATION MODELS (AQSM)

Air quality simulation models represent the most fundamental approach to relating primary pollutant emissions to secondary pollutant concentrations. This approach was first conceived and introduced by Friedlander and Seinfeld.[20] Such models are based on a mathematical description of the physical and chemical processes involved in the atmospheric behavior of air pollutants. The basis of all AQSM's is the equation of conservation of mass, which with the help of certain simplifying assumptions is reduced to equation 6-3, commonly known as the atmospheric diffusion equation:

$$\frac{\partial c_i}{\partial t} + u\frac{\partial c_i}{\partial x} + v\frac{\partial c_i}{\partial y} + w\frac{\partial c_i}{\partial z}$$

$$= \frac{\partial}{\partial x} K_H \frac{\partial c_i}{\partial x} + \frac{\partial}{\partial y} K_H \frac{\partial c_i}{\partial y}$$

$$+ \frac{\partial}{\partial z} K_V \frac{\partial c_i}{\partial z} + R_i(c_1,\ldots,c_n,T) \qquad (6\text{-}3)$$

where c_i is the mean concentration of species i; x,y,z are the Cartesian coordinates; u,v,w are the mean velocity components; K_H,K_V are the horizontal and vertical eddy diffusivities; R_i is the rate of production (or negative of the rate of consumption); T is temperature; and t is time.

The solution of the set of equations 6-3 yields the theoretical mean concentrations as a function of location and time. The solution of equation 6-3 requires, as input information, initial and boundary concentrations of each of the n species, the wind field, turbulent diffusivities, source emissions as a function of location and time (entering as boundary conditions), and a chemical reaction mechanism.

All air quality models are mathematical descriptions of the physical processes that are known to occur in the atmosphere. However, no model is a perfect descriptor; it is only as true a simulator of actual processes as the assumptions on which it is based are valid. Since models are known to be inaccurate, it is of interest to ascertain how inaccurate their predictions might be. Unfortunately, quantitative determination of the expected inaccuracy of a model is extremely difficult, if not impossible, to achieve. There are several reasons for this.

First, meteorological processes are random in nature, whereas all of the current AQSM's are deterministic (as contrasted with stochastic). Outputs (and observations) are thus meaningfully expressed in terms of random variables. Predictive results of deterministic models are not commensurate with this requirement. Thus comparison of actual observations with model predictions is not entirely appropriate. The second and related reason for the difficulty of quantitative determination is that the atmosphere is highly variable in terms of all measures used by scientists to describe it. Spatial and temporal variations of virtually all parameters that serve as inputs to models are usually quite significant and, in fact, often rather large. Third, the data base one might use both for input to the model and for comparisons with predictions is virtually always sparse and sometimes inaccurate. Finally, it is not possible to derive a totally theoretically based estimate of expected errors associated with a particular formulation, because it is not possible to formulate a truly accurate representation of the natural system with which to compare.

If equation 6-3 could be demonstrated to be an accurate representation of atmospheric processes, and if the necessary input information were known with sufficient accuracy, the AQSM would clearly be the preferred approach for relating primary emissions to secondary (e.g., oxidant) air quality. Commonly, however, there are inaccuracies associated with the use of equation 6-3. These inaccuracies can be categorized either as fundamental inaccuracies or input-related inaccuracies. The fundamental inaccuracies are those associated with the assumptions made in the derivation of equation 6-3. Such inaccuracies, for example, include those related to the assumptions made in modeling atmospheric turbulent flow. Input-related inaccuracies are those that result from uncertainties in the input information such as the source emissions, wind velocities, and the chemical reaction mechanism.

A measure of the error resulting from all sources can be obtained through comparison of the concentrations predicted by the model with those measured. Ideally, such comparisons should be made (for a particular region) for a variety of meteorological and emission conditions. One discrepancy between predictions and data will arise because equation 6-3 predicts the mean concentrations, and ambient data reflect instantaneous conditions. Also, equation 6-3 must generally be solved numerically on a grid, so that the concentration, c_i, implicitly involves some degree of spatial averaging; but the data usually consist of concentrations at a point. If a sufficient data base is available, extensive model evaluation over varying meteorological and emission conditions will provide insight into the accuracy of the model, thereby establishing the degree to which the model can be extrapolated to conditions beyond those in the domain of the evaluation.

Unfortunately, a data base that is truly suitable for model evaluation has only recently become available (see subsequent discussion), and thus conclusive model evaluation studies for AQSM for photochemical oxidant have yet to be carried out.

The subject of this section is a brief review of the capabilities and limitations of AQSM for photochemical oxidant. More extensive discussions of AQSM's can be found in several recent reviews.[15,38,51] AQSM's have the capability to predict oxidant (and other secondary pollutant) concentrations as a function of location and time over a region for any specified meteorological and emission conditions. Such prediction does require extensive data input requirements, such as a spatially and temporally resolved emission inventory and wind field, and a chemical reaction mechanism. The principal limitations associated with AQSM's result from the need for extensive input data and from inaccuracies in the input information. Because AQSM's have not yet been evaluated to the extent ultimately desired, it is difficult to provide precise indications of their ability to simulate oxidant formation accurately.

An open issue is whether or not AQSM's presently exist that are sufficiently validated and appropriate for application in designing oxidant control strategies.[14] This section outlines the key aspects of this question. First, the basic types of AQSM's for photochemical oxidant are discussed, and several specific models that are available are summarized. Then follows a discussion of the general issue of the level of detail of treatments of chemistry and meteorology in AQSM's. Finally, an attempt is made to assess the level of accuracy of AQSM's for photochemical oxidant. Most of the analysis presented here is from a recent review by Seinfeld.[51]

A synopsis of the types of AQSM's is presented in Table 6-1, and several available AQSM's for photochemical oxidant are summarized in Table 6-2. The AQSM's can be categorized according to the level of detail of the treatment of both meteorological and chemical processes. From a meteorological point of view, the basic distinction lies in the level of spatial resolution of the model. So-called grid models are based on the solution of equation 6-3 on a three-dimensional grid representing the region of interest. An example of a model of this type is the SAI model summarized in Table 6-2. There is also a class of grid models wherein explicit computations of vertical transport are not carried out; the LIRAQ model exemplifies this type. So-called trajectory models represent the chemical and vertical transport processes taking place in an advecting air column. Table 6-3 summarizes the meteorological treatments of each of the four AQSM's described in Table 6-2.

TABLE 6-1. FORMS OF AIR QUALITY SIMULATION MODELS

Form	Distinguishing features
Grid model	Model is based on numerical solution of the coupled atmospheric diffusion equations in three spatial dimensions on a grid over the region of interest.
Trajectory model	Model is based on simulating chemistry and vertical transport in air column advecting with the local mean wind velocity.
Box model	Model is based on simulating chemical processes in a well-mixed region in which no spatial inhomogeneities are assumed to exist and within which emissions are mixed instantaneously throughout the region.

With respect to chemistry, there are two types of chemical kinetic mechanisms employed in AQSM's: (1) lumped mechanisms, in which the various organic precursor species are grouped, based either on molecular structure or on reactivity, and (2) surrogate mechanisms, in which the organic species in a particular class, e.g., olefins, are represented by a single member of that class (e.g., propylene). The high degree of chemical detail in the surrogate mechanisms creates computational demands that prohibit detailed treatment of the dispersion processes. For this reason, surrogate mechanisms cannot be accommodated along with a detailed treatment of the transport process. Most AQSM's use lumped chemical mechanisms (SAI, LIRAQ, DIFKIN), whereas the Bell Laboratories model employs a surrogate mechanism. Which of these two approaches is more accurate depends on the relative errors that result from simplification of the chemical process versus the dispersion process. In his analysis of current AQSM's, Seinfeld[51] suggested that the appropriate combination of organic and free radical species in the Bell Laboratories mechanism leads to a lumped mechanism essentially similar to the lumped mechanisms in the SAI and LIRAQ models. He notes that there is virtually no advantage gained as a result of the extensive chemical detail of the Bell model at the expense of a lack of treatment of transport and dispersion in that model. Thus, models that achieve a proper balance between chemical and meteorological detail are the most appropriate for use in evaluating oxidant control strategies.

Air quality simulation models represent the preferred approach for oxidant prediction if the necessary input information for exercise of the model is available and accurate. Because the necessary input information is not always available (and may be costly to generate), and because even when it is available it may not be of an accuracy that justifies use of the model, it has been necessary to utilize empirical and semi-empirical methods for relating precursor emissions to oxidant concentrations. These methods have already been discussed earlier in this chapter. (Of course, there may be certain uses for which empirical or semi-empirical methods may be preferred over AQSM even if accurate input information for the AQSM were available. These uses would generally be those for which the cost of employing an AQSM might not be justified.)

Though considerable time and money have been devoted to the development of AQSM's for photochemical oxidant, it has generally been stated that existing models do not possess sufficient accuracy to be judged reliable. Unfortunately (but for good reason), measures of the expected inaccuracies of model predictions are rarely available. As a consequence, the utility of AQSM has been debated on a subjective rather than an objective basis. The object of the remainder of this section is, where possible, to assess the major uncertainties in AQSM and estimate the level of accuracy of oxidant predictions. (Whether, given an estimated level of uncertainty in oxidant predictions for AQSM's, the AQSM is to be preferred over another method for evaluating oxidant control strategies given an estimated level of uncertainty in oxidant predictions for AQSM depends on the levels of uncertainty of the empirical and semi-empirical methods that are alternatives.)

In deriving the atmospheric diffusion equation 6-3, the assumption of greatest concern is that of the turbulent mass flux [terms of the form $u'c_1'$, where (′) indicates the fluctuating component of velocity and concentration] being set equal to the product of a turbulent eddy diffusivity, K, and a concentration gradient. The problem is that K is not constant; rather it is a function of wind velocity, wind shear, the local vertical temperature gradient, and other variables. Thus K is a variable in time and space and is a complex function of several other

TABLE 6-2. CURRENTLY AVAILABLE AIR QUALITY SIMULATION MODELS FOR PHOTOCHEMICAL OXIDANT[52]

AQSM	Description of AQSM
SAI[a,22,29,43,44,46,48,49,52]	This three-dimensional grid model is based on numerical solution of the atmospheric diffusion equation. The three-dimensional wind field is derived from ground-level measurements. Pollutants emitted from ground-level sources are injected into the bottom layer of grid cells; emissions from stacks are distributed among the grid cells aloft. A 36-step kinetic mechanism derived from the Hecht/Seinfeld/Dodge (1974) mechanism[b,23] is used. Numerical solution is by the method of fractional steps with advection treated by the SHASTA algorithm (Boris and Book, 1973);[3] vertical diffusion and chemistry are by the Crank-Nicholson method.
LIRAQ[a,31]	This model predicts the temporal variation of pollutant concentrations in a two-dimensional array of grid cells. Each cell is bounded on the bottom and top by the terrain and inversion base, respectively. For computational purposes, the pollutants are assumed to be well mixed in each cell. An empirical algorithm is used to relate the cell-averaged concentration to the predicted ground-level value. A two-dimensional wind field is used. Pollutants emitted at ground level and aloft are injected uniformly into the appropriate well-mixed cell. A 48-step chemical reaction mechanism, similar in nature to the Hecht/Seinfeld/Dodge mechanism, is used.[c,23] The governing equations are solved using a modified version of Gear's method (Hindmarsh, 1974).[25]
DIFKIN[a,19,32]	DIFKIN is a trajectory model based on a moving column of air in which vertical diffusion and chemical reactions take place. Pollutants are emitted into the appropriate vertical cell. The column of air follows a surface trajectory interpolated from surface wind data. A 12-step kinetic mechanism is used. Governing equations are written in the form of ordinary differential equations and solved using a Pade's approximation method.
Bell Laboratory model[a,21]	This model uses three well-mixed cells in series. Wind is represented by volumetric air flow from cell to cell. Emissions are instantaneously mixed in cell. A 143-step kinetic mechanism based on detailed chemistry of propylene, formaldehyde, acetaldehyde, and propionaldehyde[d] is used. (Interaction between free radicals and aerosols is included.) Governing ordinary differential equations are solved by Gear's method (Edelson, 1976).[18]

[a] SAI = Systems Applications, Inc., model; LIRAQ = Livermore regional air quality model; DIFKIN = diffusion kinetics model.

[b] The 36-step mechanism includes five reactions describing SO_2 oxidation.

[c] The 48-step LIRAQ mechanism does not include SO_2 oxidation.

[d] The 143-step mechanism includes 19 reactions describing SO_2 oxidation.

variables. As a consequence, it is frequently quite difficult to estimate.

Solution of equation 6-3 requires specification of a wind field, (u,v,w). As the wind velocity varies in space and time, its specification, whether based on the solution of the fluid dynamics equations or on the interpolation of a set of measurements, is very difficult, and as a consequence, it is frequently one of the greatest sources of inaccuracy in model predictions. If one were to select two arbitrary locations separated by a mile or two and monitor the wind speed and direction on an hourly basis for a day, the probability is considerable that one would find significantly different readings, both in

TABLE 6-3. TREATMENT OF METEOROLOGICAL VARIABLES IN AIR QUALITY SIMULATION MODELS

Model	Advection	Mixing depth	Turbulent diffusion
SAI[a]	Model requires a three dimensional wind field. Hourly averaged surface measurements are interpolated to a fine mesh. The surface field together with the lower boundary condition, w=0, is used to derive the vertical velocities. The model employs upper level wind measurements and objective analysis procedures to render the wind-field mass consistent. Theoretical wind-shear relationships derived by Lamb[27] using the predictions of a planetary boundary layer model developed by Deardorff[6] are employed. These relationships are useful in instances when wind measurements aloft are not available. For situations in which wind measurements are available both at the surface and aloft, an objective technique for preparing appropriate three-dimensional wind inputs to the model has been developed by Reynolds.[42]	The mixing depth field is developed using areawide interpolation. Temperature profiles from radiosondes and acoustic sounder measurements are the basic inputs used to determine the depth of the mixed layer.	Vertical eddy diffusivity coefficients K_V have been derived through use of a methodology developed by Lamb et al.[27,28] In this procedure, which employs flow fields predicted by the model of Deardorff,[6] particles are released from a point and followed as they are transported downwind. From the particle trajectories, it is possible to calculate the pollutant concentration field downwind of the release point. Given the concentration and mean flow fields, the diffusivity profile is obtained through use of optimal control theory techniques. Horizontal eddy diffusivity coefficient K_H specified is an input parameter.
LIRAQ[a]	An approximate two-dimensional wind field is constructed using a Gaussian weighting function to interpolate sparse, 3-hr average wind measurements to grid cells. An iterative variational procedure is used to refine the interpolated values so as to render the field mass consistent. A feature of the technique is that it allows a parameterized treatment of flow through the inversion base and around topographic features.	Mixing depths over the region are inferred from sparse surface measurements of temperature, elevated profile data, topography, and the temperature advection from prevailing flows. These data are used to construct an interpolated field. The mixing depth is an important input parameter for the mass consistent wind-field calculation.	Spatial and temporal variations of K_H calculated are based on similarity theory. Inputs are root-mean-square dispersion, grid cell size, energy dissipation rate, mixing depth, and reference height wind velocities. Although LIRAQ is a dimensional, vertically integrated model, a value of K_V is required to establish boundary conditions for the vertical concentration profile. K_V is determined from a power-law profile for wind speed and the assumption that the friction velocity is 0.1 x the horizontal velocity at a height of 1 m.
Bell[a]	A bulk wind flow along the axis of the cells is assumed; no detailed vertical or horizontal resolution is employed in the model. Wind speed variations throughout the day are defined from the medians of seasonal observation. These data are smoothed to provide a continuous temporal wind variation.	The mixing depth is averaged over each cell, and only three spatial values are required. These data were determined from lidar measurements of vertical aerosol extent in combination with mixing height data obtained from other sources.	Diffusion in a cell is instantaneous.

[a] SAI = Systems Applications, Inc., model; LIRAQ = Livermore regional air quality model; Bell = Bell Laboratories model.

magnitude and direction, between the locations. Thus an accurate model must allow for the possibility of a laterally varying wind field. In addition, variations of wind speed with height can often be substantial, so it is necessary that a model include wind shear. As noted, the problems associated with determining the spatially and temporally varying wind field for a particular time period in an area are substantial. Measurements of winds aloft are typically unavailable, and except for special circumstances, it is very difficult to predict wind speed and direction at elevations much removed from the surface, with only ground observations available. In addition, the density of ground-level wind stations is generally less than that required to interpolate a ground-level wind field with a resolution comparable to that of the AQSM.

The chemical mechanism is that element of the AQSM that has undergone the most change over the period of time that models have been developed. This change has been the result of continuous investigation of the chemistry of photochemical smog and includes revised rate constants and new products for some of the individual reactions. Whereas the chemical mechanisms employed in various models differ (see Table 6-2), an increasing degree of uniformity in the treatment of the chemical processes is beginning to emerge as new chemical information is incorporated into the mechanisms in the AQSM's. The most direct evidence of the adequacy of a chemical mechanism is that obtained through simulation of smog chamber data.

In principle, every reaction appearing in a photochemical smog mechanism is subject to some degree of uncertainty, whether in the rate constant or the nature and quantity of the products. In validating a mechanism, the accepted procedure is to compare the results of smog chamber experiments, usually in the form of concentration-time profiles, with simulations of the same experiments using the proposed mechanism. A sufficient number of experimental unknowns exist in all such mechanisms that a certain degree of adjustment of rate constants (and perhaps products) is possible. The inherent validity or accuracy of any mechanism should be judged on the basis of how realistic the parameter variations are.

Uncertainties in the kinetic mechanism are related to inaccurately known rate constants or products for reactions in the mechanism. Uncertainties associated with comparison of the predictions of the mechanism to experimental smog chamber data arise, in addition, because the properties of the photochemical reactor, associated equipment, and experimental procedures are not completely known. The accuracy of any method based on smog chamber data for relating precursor emissions or concentrations to oxidant levels, (whether it is based directly on ozone isopleths as a function of initial precursor concentrations or on a kinetic mechanism validated with smog chamber data) depends on the extent to which the influence of the smog chamber on the homogeneous kinetics is understood. Some of the specific chamber effects that must be considered are the spectral distribution and absolute intensity of the photolyzing lamps; the adsorption, desorption, and chemical reaction of species on the walls; the initial loading of impurity species in the chamber air or on the walls; and the effects of leakage, sampling, and possible temperature variations during the run. Of these effects, probably the most important are the effects of the photolyzing lamps and of species absorbed on the walls. Photolytic rates of absorbing species cannot be predicted with accuracy if the incident light intensity distribution is not known with accuracy. This information must be coupled with the absolute rate of photolysis of at least one species, such as NO_2, in order to compute the appropriate photolytic rate constants. Characterization of the initial contaminant loading in the gas and on the walls is important in simulating the proper initial rates of conversion.

In summary, the three classes of phenomena that often require treatment as parameters in kinetic mechanisms in simulating smog chamber data are: (1) unknown rate constants, (2) the photolytic properties of the reactor, and (3) wall absorption, desorption, and heterogeneous chemistry. Tuning the mechanism to account for unknown or uncertain chemical and physical effects is a legitimate procedure provided that the exact steps are spelled out in detail and lie within physically realistic bounds. Recent experimental elucidation of NO_x/HO_x chemistry (reactions of OH and HO_2 with NO and NO_2) and PAN chemistry has significantly improved the accuracy of postulated mechanisms for photochemical air pollution. There still exist, however, important uncertainties in RO_x/NO_x chemistry (reactions of RO and RO_2 with NO and NO_2). Nevertheless, a properly tuned mechanism is capable of predicting the concentration-time profiles of species such as NO, NO_2, ozone, and hydrocarbons within 20 percent over a wide range of initial conditions. In theory, such a mechanism, minus the steps included to account exclusively for chamber effects, should be capable of predicting atmospheric concentrations with the same accuracy.

Ozone prediction uncertainties due to uncertainties in the meteorological and emission inventory input data have been explored in a comprehensive sensitivity study by Liu et al.[30] The uncertainties in predicted ozone concentrations from all mechanisms and input uncertainties were estimated by Seinfeld[51] (Table 6-4). These estimates from the evaluation studies illustrate the magnitudes of the possible errors associated with the various components of an AQSM. The total error, of course, cannot be determined from such data alone. Seinfeld did estimate an overall uncertainty value of ± 50 percent.

TABLE 6-4. UNCERTAINTIES IN PREDICTED OZONE CONCENTRATIONS FROM ALL MECHANISMS AND INPUT UNCERTAINTIES

Source	Uncertainty in predicted absolute ozone concentrations, %
Chemical mechanism	±20
Meteorology:	
Wind speed and direction	±20
Mixing depth	±25
Light intensity	±20
Initial and boundary conditions	±50
Emission inventories:	
NO_x	±20
Hydrocarbons	±30

Evaluation studies using some of the current AQSM's are summarized in Table 6-5. As shown, the variety of regions and conditions for which the AQSM's have been evaluated is limited. In particular, there is a lack of evaluations under widely varying emission conditions. Most important, however, evaluation periods have been limited to 1 to 3 days, or 6 days at the most. Overall, the past evaluations are inadequate in several respects. First, because of deficiencies in the data bases used, it is difficult to discern whether or not the disagreements between prediction and observations are the result of errors in the model, inaccuracies in input parameters, or deviations arising from comparison of point data and volume-average prediction. Second, the variety of conditions used in the verification tests was limited, as already mentioned. Third, and perhaps most important, the amount of verification in terms of number of test days is limited to the extent that the resulting assessment of the model's accuracy is qualitative, at best; certainly, it is not sufficient for statistically supported quantitative estimates. And finally, another problem in interpreting results from current verification studies is that of tuning of model parameters or inputs (adjustment of some influential parameters within their uncertainty limits to maximize agreement between predictions and observations). Such tuning varies in extent from study to study. However, even in the most serious studies, there are required data (e.g., initial and boundary concentrations aloft) that are simply not available and must be estimated; such input data are often adjusted to obtain the best fit. Thus the value of the model predictions is lessened to an extent, depending on the amount of tuning performed and, more important, on the sensitivity of the model to the parameters tuned.

A major verification study involving several of the current AQSM's is now close to completion and has been reported extensively.[15] Known as the St. Louis Regional Air Pollution Study (RAPS), this study is a 5-year field program sponsored by EPA. The major objective is the development, evaluation, and verification of AQSM's. The magnitude of the study is such that the amount of data to be obtained will be adequate for a statistical assessment of AQSM performance. Thus some 50 simulation days will be considered for statistical evaluation of each model. The statistical procedures to be used will be based on methods such as those reported by Brier,[4] Nappo,[36] and Liu et al.[30] Following completion of this major effort, and based on AQSM performance standards to be defined, EPA will make judgments on the specific AQSM's to be recommended for use in the development of oxidant control strategies.

SUMMARY

Quantitative relationships between ambient oxidant/ozone and precursor emissions are needed for predicting the impact of emissions on air quality. Such relationships represent, with varying degrees of complexity, the physical and chemical processes taking place in the atmosphere.

The most fundamental approach to relating precursor emissions to oxidant air quality is that based on the equation of conservation of mass, which with the help of certain simplifying assumptions, is reduced to the atmospheric diffusion equation (equation 6-3). This approach, which uses AQSM's, requires knowledge of the wind field and boundary conditions of the region, the source emissions as a function of location and time, and a chemical reaction mechanism. The complexity of air quality simulation models (AQSM) is a source of both strength and weakness. The high degree of spatial and temporal detail makes the models suitable for a wide variety of applications, but at the expense of costly computations. No AQSM is a perfect description of the atmosphere; inaccuracies arise from assumptions needed to represent atmospheric turbulent processes in a manageable form and from uncertainties in the input information, such as chemical reaction rate constants. The largest sources of uncertainty in model predictions are caused by uncertainties in the initial and boundary conditions and in the chemical mechanism. Uncertainties are introduced also by errors in the wind, mixing height, and light intensity, and in the HC and NO_x emission inventory data. To date, verification of existing AQSM's has been inadequate in at least two respects. First, because of deficiencies in the data base used, it has been difficult to discern whether the disagreements between observations and model predictions are the result of errors in the model, inaccuracies in input parameters, or deviations arising from comparison of point data and volume-average prediction. Second, the amount of validation in terms of number of test days and the variety of conditions used in such validation have been limited to the extent that resulting assessments of model accuracy have been, at best, qualitative.

Because AQSM's have not been developed to the point that their degree of accuracy is well-quantified, it has been necessary to employ empirical and semi-empirical methods for relating precursor emissions to oxidant air quality. Of the empirical methods for relating precursor emissions to oxidant air quality, linear rollback is the simplest; it is based on assumed proportionality between ambient oxidant/ozone concentration and the reactive organic emission rate. The method is of highly questionable validity and limited utility, since most information indicates that peak oxidant concentrations do not decrease linearly with reductions in hydrocarbon emissions.

TABLE 6-5. PRIOR VALIDATION STUDIES OF AIR QUALITY SIMULATION MODELS

Model	Region	Time periods	Pollutants compared
SAI[a]	Portions of south coast air basin. Both 50 x 50 mi and 80 x 100 mi regions. Denver, Colo. (SAI, 1977).	6 days in 1969 26 June 1974[41,45]	Validation studies performed with both the 15- and 31- step kinetic mechanisms. In both versions, pollutants compared were NO, NO_2, O_3, CO, reactive and unreactive hydrocarbons.
LIRAQ[a]	San Francisco Bay area. 170 x 210 km region. A variety of subregions and grid sizes (1-5 km) were employed.	26, 27 July 1973[31] 20 August 1973[31] 26-28 September 1973[31]	Pollutants compared were NO, NO_2, O_3, CO, reactive and unreactive hydrocarbons.
DIFKIN[a]	Trajectories in south coast air basin.	6 days in 1969 LARPP data [53]	Pollutants compared were NO, NO_2, O_3, CO, reactive and unreactive hydrocarbons.
Bell[a]	Morris, Essex, Hudson counties, New Jersey (Comparisons shown only for Hudson County).	May-September 1972-1974. Cloudless summer weekdays with normal convective mixing and westerly winds. Only days when the integrated 0600-1300 hr solar flux was greater than 200 Langleys, average 0400-1300 hr wind velocity 6.7 mi/hr $\leq v \leq$ 10.1 mi/hr, and 0400-1300 hr wind direction was from 80° sector encompassing Essex County to the west-northwest.[21]	O_3, NO, NO_2, SO_2 and computed O_3 behavior matches well the median of data. Computed concentration levels of secondary species agree qualitatively with levels measured in New Jersey or elsewhere (acrolein, acetaldehyde, formaldehyde, PAN, H_2O_2, HNO_2, HNO_3). Levels of HO_2 radicals estimated on the basis of predicted H_2O_2 levels. Levels of OH radicals estimated on the basis of predicted HNO_3 levels.

[a] SAI = Systems Applications, Inc.; LIRAQ = Livermore regional air quality; DIFKIN = diffusion kinetics; Bell = Bell Laboratories.

The "Appendix-J method" is a modified version of linear rollback in which ambient oxidant/ozone is related to hydrocarbon precursors in the form of an upper limit curve derived from aerometric data from several cities on 6- to 9-a.m. NMHC and maximum oxidant concentration. The method is no more valid than linear rollback, except perhaps when used for one locality on the basis of data taken in that locality. Statistical methods that relate ambient oxidant/ozone to both HC and NO_x precursors are superior to linear and modified rollback methods that are based only on hydrocarbon precursors. The methods, however, should be employed only for the locality from which the data on which they are based have come.

An attempt to base the precursor/ozone relationship on chemical reality while avoiding the complex computational requirements of AQSM led to the development by Dimitriades of peak oxidant isopleths as a function of initial hydrocarbon and NO_x concentrations derived from smog chamber data. The advantage of the approach is that it is based on actual smog chamber data and also on the results of simulations with a chemical reaction mechanism validated with smog chamber data. The limitations of the method arise from lack of spatial and temporal resolution and from the questionable comparability of the atmosphere of a smog chamber with the real atmosphere.

In summary, air quality simulation models represent the most fundamental approach to relating precursor emissions to oxidant air quality. Although AQSM's have now evolved to the point where they can be employed with some confidence, they are still characterized by uncertainties that arise from our lack of ability to represent truly atmospheric processes. Based on extensive evaluation of several AQSM's with the St. Louis RAPS data, EPA will make judgments on specific AQSM's to be recommended. Because of their extensive computational requirements, even where fully validated, the AQSM approach may not be suitable for all applications involving oxidant control planning.

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7. MEASUREMENT METHODS FOR OZONE, OXIDANTS, AND THEIR PRECURSORS

INTRODUCTION

Since the publication of the air quality criteria documents for photochemical oxidants,[60] hydrocarbons,[61] and nitrogen oxides,[1] there have been significant advances in the measurement of these pollutants in ambient air. The chemiluminescent reaction of O_3 with ethylene has been successfully exploited to produce instrumentation whose response is specific for O_3 and is linear with O_3 concentrations over the range found in ambient air. Advances in electronics technology have allowed development of photometers of adequate precision to measure O_3 by ultraviolet absorption photometry. Continuous analyzers based on the chemiluminescent reaction of NO with O_3 have been successfully developed for the measurement of NO, nitrogen dioxide (NO_2), and oxides of nitrogen (NO_x). Improved manual (integrated) methods for measuring NO_2 have also been developed. Lesser advances have been reported in the measurement of ambient hydrocarbons and peroxyaclynitrates.

The emphasis in this chapter will be placed primarily on methods for measuring O_3, on the O_3/oxidant relationship, and on methods for measuring hydrocarbons and NO, NO_2, and NO_x. Continuous methods for measuring total oxidants based on the reaction of ambient air oxidants with potassium iodide (KI) will also be discussed briefly. A more detailed discussion of total oxidant methodology is given in the earlier criteria document.[60]

SAMPLING FACTORS IN AMBIENT AIR MONITORING

In addition to analytical principles and measurement procedures, sampling factors also have a crucial effect on the quality of ambient and experimental atmosphere measurements. In sampling the ambient atmosphere, it is extremely important that the sampling be performed in a manner that (1) is consistent with the specific purpose of the measurement, and (2) preserves the integrity of the pollutant mixture in the ambient air sample. These sampling factors and their significance in O_3 or oxidant-related air quality monitoring will be discussed here briefly. For more detailed discussions of this subject, the reader is referred to other EPA documents and to reports prepared for EPA by the National Academy of Sciences.[62,63,67,72]

Air monitoring data relevant to the ambient O_3 or oxidant problem are collected for a diversity of specific purposes, including serving as an indicator of progress in attainment of the national ambient air quality standard for O_3 in the development of O_3 control strategies, in the development and validation of ozone-related air quality simulation models, in the investigation of causes of ozone problems, etc. Each of these specific purposes or needs requires special considerations in designing an air sampling strategy responsive to the respective need. For example, statistical considerations enter into the design of a monitoring network capable of detecting air quality changes over a given period of time. Other considerations arising from the chemical, physical, and meteorological aspects of the ambient ozone problem are explained briefly as follows.

Ozone is a product of photochemical reactions that involve sunlight, hydrocarbon, and NO_x reactants, which are heavily discharged into urban atmospheres by automobiles during the morning peak traffic hours. This photochemical process occurs at a rate such that the O_3 concentration reaches its daily peak level some time in midday at locations downwind from the source-intensive center-city area. Thus if peak O_3 concentrations are to be measured, monitoring stations should be located downwind from city centers at distances that have been determined by EPA to be 15 to 30 km (9 to 19 miles), depending on the area's predominant wind patterns.[67] Monitoring stations should also be located within the source-intensive area for measurement of peak concentrations of oxidant precursors. Once a station is located, additional considerations arise because of the chemical reactivity and instability of the O_3 molecule. Ozone reacts extremely rapidly with NO and with some hydrocarbon compounds, including most of those emitted by vegetation. Also, O_3 decomposes readily on contact with the surface of many materials. Consideration of these effects led to the development of specific criteria for locating an O_3 monitoring station.[62,63,67,72] Briefly, the inlet of the O_3 analyzer's sampling probe should be positioned 3 to 15 m (10 to 49 ft) above ground, at least 4 m (13 ft) from large trees, and 120 m (394 ft) from heavy automobile traffic. Sampling probes should be designed to minimize O_3 destruction by surface reaction or by reaction with NO.

Air monitoring data, as commonly obtained, have only limited validity as measures of absolute air quality. The reason is that at ground level, the ambient atmosphere is inhomogeneous as a result of a continuous influx of fresh emissions, incomplete mixing, and destruction of O_3 by fresh and unreacted emissions. In view of such inhomogeneity, monitoring data from a fixed network provide measures of air quality at a discrete number of locations but may not detect possibly existing hot-spots. This problem can be alleviated by use of a greater density of monitoring stations or, perhaps at a lower cost, by use of an air quality model. Such models (Chapter 6) are capable of quantifying the emission dispersion and chemical reactions processes, and their outputs can provide data on the distribution of air quality concentrations between widely spaced ambient monitors.

MEASUREMENT OF OZONE

Gas-Phase Chemiluminescence (EPA Reference Methods)

The U.S. Environmental Protection Agency promulgated National Ambient Air Quality Standards (NAAQS) for six pollutants on April 30, 1971.[104] The standards are now codified at Title 40, Code of the Federal Register (40 CFR), Part 50. At the same time, EPA published reference methods (presently described in the appendices to Part 50) to be used by EPA and by state and local agencies in measuring ambient concentrations of the six pollutants. Appendix D of 40 CFR Part 50 for the measurement of photochemical oxidants describes the measurement principle for an automated method (analyzer) based on the gas-phase chemiluminescent reaction of O_3 with ethylene.[65,99] Measurements made with analyzers based on this principle are therefore ozone-specific and differ from measurements made with analyzers designed to measure total oxidant.

These analyzers are designed such that ambient air and ethylene are delivered simultaneously to a reaction cell where the O_3 in the air reacts with the ethylene. The reaction produces a small fraction of an energetically excited species (thought to be an electronically excited formaldehyde molecule) that decays to the ground state with the emission of light. The intensity of the emitted light (chemiluminescence), which is detected by a photomultiplier tube, is proportional to the O_3 concentration over the range of 4 to at least 5000 $\mu g/m^3$ (0.002 to at least 2.5 ppm). The quantitative relationship between the intensity of the chemiluminescence and the O_3 concentration must be established for each analyzer, using atmospheres containing known concentrations of O_3.

Analyzers utilizing the gas-phase chemiluminescence measurement principle have been evaluated under a variety of conditions.[7,18,22,98] These studies indicate that the performance of commercially available analyzers is generally satisfactory. However, the results of a collaborative test by McKee et al. indicated considerable variability and a negative bias of 16 to 37 percent.[58] The reason for the variability is no doubt, in part, the variability in the 1 percent neutral buffered potassium iodide (NBKI) calibration procedure, which is discussed in detail in a subsequent section on O_3 calibration procedures. The reported negative bias is in conflict with subsequent studies of the 1 percent NBKI procedure, which generally indicate that this procedure produces a positive bias of 5 to 30 percent (see section on O_3 calibration procedures).

Under the provisions of the Ambient Air Monitoring Reference and Equivalent Methods Regulations[102] promulgated February 18, 1975 (40 CFR Part 53), several commercial analyzers have been designated as reference methods for determining compliance with the NAAQS for photochemical oxidants. These analyzers have been subjected to the required testing and have met the EPA performance specifications for automated methods. These specifications are given in Table 7-1, and a list of the ozone analyzers (designated as of March 3, 1978) is given in Table

7-2. Information concerning the applications supporting the designation of these analyzers as reference or equivalent methods may be obtained by writing the Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711.

TABLE 7-1. PERFORMANCE SPECIFICATIONS FOR AUTOMATED METHODS

Performance parameter	Ozone
Range, ppm	0-0.5
Noise, ppm	0.005
Lower detectable limit, ppm	0.01
Interference equivalent:	
Each interferant, ppm	±0.02
Total interferant, ppm	0.06
Zero drift, 12- and 24-hr, ppm	±0.02
Span drift, 24-hr:	
20% of upper range limit, ppm	±20.0
80% of upper range limit, ppm	±5.0
Lag time, min	20
Rise time, min	15
Fall time, min	15
Precision:	
20% of upper range limit, ppm	0.01
80% of upper range limit, ppm	0.01

A review of the performance data submitted in support of the designations listed in Table 7-2 indicates that these analyzers exhibit performance better than that specified in Table 7-1. For the analyzers tested, the zero drift results (12- and 24-hr) were all less than 5 ppb and typically less than 3 ppb. The span drift results (at 20 and 80 percent of the full-scale range of 0 to 0.5 ppm) were all less than 5 percent, and typically 2 to 3 percent. The precision results (at 20 and 80 percent of the full-scale range of 0 to 0.5 ppm) indicate a typical precision of 1 ppb. The response times (lag, rise, and fall) were all less than 2 min, and typically less than 1 min. The interference equivalent results (tests for carbon dioxide, hydrogen sulfide, and water vapor) were typically less than 1 ppb. However, under 40 CFR Part 53, the test procedure for water interference specifies that the test be conducted in the absence of O_3. There have been a number of reports of a positive water interference of 3 to 12 percent under conditions of high humidity in the presence of O_3.[51,53,73]

Gas-Solid Chemiluminescence

The measurement of O_3 by a gas-solid chemiluminescence technique was first reported by Regener.[77,78] In Regener's approach, air containing O_3 is passed across a surface of Rhodamine B and absorbed on silica gel, resulting in the emission of light (chemiluminescence). The intensity of the emitted light is measured with a photomultiplier tube and is proportional to the concentration of O_3 in the air sample. The method is highly specific and extremely sensitive (lower detection limit less than 0.001 ppm). Because the sensitivity of the chemiluminescence surface gradually decays with time, automated analyzers based on this measurement principle are designed to incorporate frequent internal calibration cycles. Prototype Regener O_3 analyzers have been evaluated under field conditions and have given excellent performance.[6]

A commercial analyzer, based on a slight modification of the gas-solid chemiluminescence principle, has been designated as an equivalent method under the EPA 40 CFR Part 50 regulations (Table 7-3). This analyzer, the Philips Model PW9771 O_3 Monitor, uses a detector disc containing Rhodamine B and an intermediate reagent, gallic acid. The O_3 in the air sample reacts with the gallic acid to produce oxygen and a gallic acid derivative, which in turn reacts with the Rhodamine B to produce the chemiluminescence.

Ultraviolet Photometry

Older ozone monitors, based on the absorption of ultraviolet (UV) light, and their attendent problems are described in the earlier criteria document for photochemical oxidants.[60] Modern electronic techniques have permitted the successful development of an ambient O_3 analyzer based on ultraviolet absorption.[12] This analyzer, the Dasibi Model 1003-AH Ozone Analyzer, has been designated as an equivalent method under the EPA 40 CFR Part 53 regulations (Table 7-3). The absorption of 254 nm ultraviolet light by O_3 in an ambient air sample is measured, and a separate measurement is made on a similar air sample from which the O_3 has been removed by a manganese dioxide (MnO_2) scrubber. These two measurements are processed electronically to produce a digital readout of the O_3 concentration in the air sample. The O_3 determination is based on the well established absorption coefficient of O_3 for 254 nm light.[23,32,36,43,100]

Although not in wide use, this method is used extensively in California. Its main advantage is that it does not require the use of support gases such as ethylene (as is the case with the gas-phase chemiluminescence methods). In addition, the O_3 measurement can be referenced to the absorption coefficient of O_3 without reference to a dynamic calibration standard. In practice, however, analyzers may require dynamic calibration with O_3 standards because of problems associated with sample integrity. While potential interferences are few, there are some ambient pollutants, including sulfur dioxide and benzene, that absorb light at 254 nm. Presumably, these potential interferants are unaffected by the MnO_2 scrubber and therefore do not interfere with the ozone measurement. However, if the MnO_2 scrubber eliminates or reduces such components, the analyzer gives erroneous readings. Field use of this analyzer is discussed by Zafonte et al.[104]

MEASUREMENT OF TOTAL OXIDANTS

The methods most frequently used in the past for measurement of ambient total oxidants were based on the oxidation of potassium iodide (KI) in solution and the electrochemical or colorimetric detection of the iodine (I_2) produced. The KI reagent produces I_2 by reaction with O_3, NO_2, peroxyacylnitrates, chlorine (Cl_2), bromine (Br_2), and peroxides.[40,85,101] Reducing agents such as SO_2 are negative interferants because they react with the I_2 formed by the oxidizing species. Terms such as "corrected oxidant" or "adjusted oxidant" have often been used to indicate that the oxidant measurement has been corrected for interferences from NO_x and/or SO_2. The corrected or adjusted measurement results are then taken to represent an oxidant mixture that is predominantly O_3 with small amounts of other oxidizing compounds.

TABLE 7-2. LIST OF DESIGNATED REFERENCE METHODS

Designation number	Identification and source	Fed. Register Notice		
		Vol	Page	Date
RFOA-1075-003	Meloy Model OA 325-2R Ozone Analyzer Meloy Laboratories, Inc. 6715 Electronic Drive Springfield, Virginia 22151	40	54856	11/26/75
RFOA-1075-004	Meloy Model OA 350-2R Ozone Analyzer Meloy Laboratories, Inc. 6715 Electronic Drive Springfield, Virginia 22151	40	54856	11/26/75
RFOA-1076-007	Bendix Model 8002 Ozone Analyzer The Bendix Corporation Post Office Drawer 831 Lewisburg, West Virginia 24901	41	5145	2/4/76
RFOA-1076-014	MEC Model 1100-1 Ozone Meter	41	466747	10/22/76
RFOA-1076-015	MEC Model 1100-2 Ozone Meter		30235	6/13/77
RFOA-1076-016	MEC Model 1100-3 Ozone Meter Columbia Scientific Industries 11950 Jollyville Road P.O. Box 9908 Austin, Texas 78766			
RFOA-1176-017	Monitor Labs Model 8410E Ozone Analyzer Monitor Labs, Inc. 4002 Sorrento Valley Blvd. San Diego, California, 92121	41	53684	12/8/76
RFOA-0577	Beckman Model 950A Ozone Analyzer 2500 Harbor Boulevard Fullerton, California 92634	42	28571	6/3/77

TABLE 7-3. LIST OF DESIGNATED EQUIVALENT METHODS

Designation number	Identification and source	Fed. Register Notice		
		Vol	Page	Date
EQOA-0777-023	Philips PW9771 O_3 Analyzer, Philips Electronic Instruments, Inc. 85 McKee Drive Mahwah, New Jersey 07430	42	38931	8/1/77
EQOA-0577-019	Dasibi Model 1003-AH Analyzer Dasibi Environmental Corp. 616 E. Colorado St. Glendale, California 91205	42	28571	6/3/77

A variety of KI methods have been used for the continuous measurement of ambient total oxidants. Amperometric[85,101] (often called coulometric) analyzers draw the air sample into a sensor cell where the oxidant reacts with the electrolyte (neutral buffered KI:KBr solution) to release I_2. The I_2 is in turn reduced back to iodide, either electrolytically with a bias voltage across the cell, as used by mast, or galvanically, with different cell electrode materials according to Hersh and Deuringer.[37] Nitrogen dioxide is a positive interferant that causes a response equivalent to 3 to 20 percent of that of O_3, depending on a detector design, operating conditions, and other unknown factors. Sulfur dioxide results in a negative interference if I_2 is present in the electrolyte. When oxidant is present in excess, a given concentration of SO_2 reduces the oxidant response on a mole O_3/mole SO_2 basis.

Colorimetric analyzers utilize either a 10- or a 20-percent neutral buffered KI[40,60,101] solution. Oxidants react with KI to produce I_2 and the triiodide ion (I_3), which is formed in the presence of a large excess of iodide ion and is measured in a continuous colorimeter. Sulfur dioxide results in a negative response similar to the case for the amperometric analyzer. Nitrogen dioxide causes a positive response equivalent to 21 and 30 percent of that of O_3 for the 10- and 20-percent KI analyzers, respectively.

To eliminate errors resulting from SO_2 interference with these KI methods, it is necessary either to measure SO_2 and adjust the total oxidant measurement appropriately, or to remove SO_2 by passing the air sample through a chromium trioxide (CrO_3) scrubber.[40] In the first case, adjustments can be made only if the oxidant levels are in excess of the SO_2 levels. In the latter case, the CrO_3 scrubber may cause new interferences by partially oxidizing the nitric oxide (NO) and hydrogen sulfide (H_2S) possibly present in the sample to NO_2 and SO_2, respectively, and by destroying part of the oxidant. Because of these potential problems, this method of correcting for SO_2 interference can be used reliably only in certain situations and only by skilled analysts.[63,85,101]

OZONE CALIBRATION PROCEDURES

Calibration of O_3 analyzers is complicated by the lack of Standard Reference Material (SRM) for O_3 analogous to those available from and certified by the National Bureau of Standards (NBS) for SO_2, NO, and carbon monoxide (CO). The instability of O_3 prohibits the storage of O_3 standards for any practical length of time. Therefore, standard samples in air for calibration of O_3 analyzers must be generated and analyzed at the time and place of use. Typically, O_3 atmospheres are generated by means of an O_3 generator that delivers a stable concentration of O_3. These atmospheres are assayed by some technique to determine their O_3 concentration and then are used to calibrate the analyzer. The O_3 assay procedure may be based on one of several different primary standards.

KI Calibration Procedures

Until recently, O_3 and total oxidant analyzers have been calibrated using O_3 atmospheres that have been assayed using various KI procedures. Such procedures, however, have been severely criticized lately for lack of accuracy and precision.[9,15,24,101] Some specifics of these procedures and associated criticisms are briefly reviewed here.

The determination of the precise O_3 atmospheres by the KI procedures consists of bringing the air/O_3 mixture into contact with buffered or unbuffered KI solutions of various concentrations. In theory, absorption of one molecule of O_3 in any of these solutions should result in formation of one molecule of I_2 as shown by the following equation:

$$2H^+ + 2I^- + O_3 = I_2 + O_2 + H_2O$$

The I_2 in the presence of a large excess of iodide ion forms the intensely colored triiodide ion (I_3^-). The concentration of I_3^- is determined using a spectrophotometer calibrated with standard solutions of I_2/KI, standardized against a primary standard such as arsenous oxide (As_2O_3). The most commonly used procedures utilize a l- or 2-percent NBKI solution, or a 2-percent unbuffered KI(UKI) solution. When EPA first promulgated the regulations (40 CFR Part 50) on April 30, 1971,[104] the 1-percent NBKI procedure was considered to be the best available for calibration of methods for measuring photochemical oxidants. This procedure is described in Appendix D of 40 CFR Part 50.

Considerable evidence has accumulated to indicate that the use of NBKI calibration procedures can result in significant bias, variability, or both. These problems were documented in a joint EPA-NBS workshop held in August 1974.[19] Specific problem areas reported included purity of reagents, time for maximum color development, variability due to impinger type, and a positive bias when compared to other ozone measurement methods such as UV photometry. An intercomparison of three KI procedures was carried out in California for the purpose of determining the relative (accuracy) responses and precisions of the three techniques. The three techniques were the 1-percent NBKI procedure (as used in the EPA reference method) the 2-percent NBKI procedure (as used by the California Air Resources Board), and the 2-percent UKI procedure (as used by the Los Angeles Air Pollution Control District). The absolute levels of O_3 were measured using UV photometry. The comparative results are fully described in a report by DeMore et al.[24] and should be consulted for details. The major conclusions of the study are that the EPA (1-percent NBKI) and California Air Resources Board (2-percent NBKI) procedures give higher values by about 15 to 25 percent compared to the UV procedure. The Los Angeles County Air Pollution District procedure (2-percent UKI) gives values approximately 4 percent lower than the UV photometry measurements, but with considerable scatter in its values. Several other studies have indicated discrepancies and considerable variability between the various KI procedures and newly developed procedures based on GPT and ultraviolet absorption (UV).[9,15,39,41,51,53,73,79] All of these and other studies have been reviewed recently by Burton et al.[14]

Based on the results of the studies discussed or cited above, it can be concluded that the KI procedures for measuring O_3, even when used in the laboratory by skilled operators, suffer from substantial systematic (accuracy-related) as well as random (precision-related) errors. The factors responsible for these errors, especially the random errors, are not well understood. They are probably related to differences in procedural detail among the various operators. When these KI procedures are used in the field by unskilled operators, such errors are certain to be considerably greater. The most important implication of these problems with the KI procedures is discussed later in the section on the relationships between ambient oxidant and ozone data.

Recently Developed Calibration Procedures

There has been a sustained effort to develop other techniques for measuring absolute concentrations of ozone in calibration atmospheres. These efforts have been successful, and it is all but certain that the KI calibration procedures will soon be replaced by one or more new techniques.

On October 6, 1976, EPA published in the *Federal Register*[103] a notice of its intent (1) to study and evaluate several alternative calibration procedures for reference methods for photochemical oxidants and (2) to amend Appendix D of 40 CFR Part 50 to revise the 1-percent NBKI calibration procedure or to replace it with one or more alternative procedures. Four alternative calibration procedures are currently under consideration: (1) GPT with excess NO, (2) UV photometry, (3) GPT with excess O_3, and (4) boric acid KI (BAKI). A brief summary of each of these alternative procedures follows.

The O_3 calibration procedure utilizing GPT with excess NO is a relatively well developed procedure and very similar to that specified by EPA for calibration of NO_2 reference method analyzers.[105] The procedure is based on the reaction of O_3 with a known quantity of NO and uses available NO standards that can serve double duty in generating NO_2 standards as well. However, an NO analyzer, which may not be readily available when calibrating O_3 analyzers, is required. In addition, the procedure is somewhat complex and requires accurate measurement of several gas flow rates.

The UV photometry procedure is based on the absorption coefficient of O_3 at 254 nm, which has been well established by independent determinations. The procedure is quite easy to carry out, uses a physical measurement, and requires no other gases or critical flow measurements. However, the procedure as applied to the calibration of O_3 analyzers is relatively new and not in general use in the air monitoring community and has not been used extensively to measure O_3 in the sub-ppm range needed for ambient O_3 analyzer calibration.

The procedure utilizing GPT with excess O_3 is similar to GPT with excess NO but has the advantage of not requiring an NO analyzer. If the residence time of the O_3 and NO reactants in the GPT reaction chamber is not carefully controlled, errors can result from either incomplete reaction of NO or reaction of the resulting NO_2 with residual O_3. For this reason, accurate use of the procedure is difficult, and further development may be required before it is recommended for use under field conditions.

Boric acid KI is a modification of the NBKI calibration procedure. This procedure utilizes a 1-percent KI solution acidified with 0.1 molar boric acid. Preliminary results indicate that this modification vastly improves both the variability and accuracy of the KI procedure.[27]

RELATIONSHIPS BETWEEN AMBIENT OXIDANT AND OZONE DATA

The relationship, and more specifically the direction and magnitude of differences, if any, between chemiluminescence O_3 (chemilum-O_3) data and KI data taken in parallel from ambient atmospheres are of interest because they relate to the issue of the definition and justification of the numerical air quality standard for photochemical oxidants.[26] This issue arises from uncertainty regarding the specific species responsible for the health effects attributed to photochemical oxidants. The problem seems to be whether the epidemiological evidence or the ozone-specific toxicological evidence should be used as the main basis of the numerical air quality standard. Thus, for example, if the epidemiological evidence alone were selected as the main basis for the air standard, then the responsibility for the observed health effects would be placed on the entire photochemical pollution complex rather than on specific constituent(s), since epidemiological evidence is not pollutant specific. In that case, the smog constituent(s) measured and correlated with the epidemiological evidence (i.e., oxidants determined by KI) would be viewed as a surrogate species. This, in turn, introduces two requirements. First, the numerical air quality standard must be defined either in terms of the same group of oxidants that was correlated with the epidemiological evidence (i.e., oxidants determined by the KI method) or in terms of an equivalent species (e.g., oxidants determined by the chemilum-O_3 method). An equivalent species is one present at concentrations equal to those determined by the KI method for measurement of oxidants. Second, the composition of the photochemical oxidants mixture, or at least the ratio of oxidants determined by the chemilum-O_3 method to oxidants determined by the KI method should be constant with location and time.

In contrast to the preceding case, if toxicological evidence is used alone or in conjunction with epidemiological evidence to place the responsibility for the health effects on a specific species, O_3 for example, then the numerical air quality standard should be defined in terms of O_3, and the correlation or lack of correlation between O_3 and other smog constituents could be disregarded as being irrelevant.

The question of whether the air quality standard should be defined in terms of oxidant-by-chemilum-O_3, oxidant-by-KI, or any other measurable entity will not be examined here. The comparison, however, of oxidant data obtained by the KI method and by the chemilum-O_3 is relevant and will be discussed briefly.

As discussed in the previous section on KI calibration procedures, the experiences from the laboratory application of KI procedures have demonstrated that such procedures, whether used in standardizing ozone mixtures or in measuring ambient oxidant, are substantially imprecise and inaccurate because of the effects from factors that are not completely understood. When the same standard O_3 mixtures are used to calibrate a KI method and the chemilum-O_3 method, then, obviously, any difference in ambient measurements obtained by the two methods reflects errors other than those related to calibration. Such errors include: (1) systematic errors related to the different response specificities of the two methods; (thus, for example, the KI method should give higher results because it responds to more oxidants than the chemilum-O_3 method), and perhaps more important, (2) disagreement between the two methods will reflect the unknown-origin random errors discussed in the preceding section. Because of the random nature of these latter errors, and because of their connection to the operator factor, the true difference in results between two methods cannot be determined from a single day's side-by-side comparison of the two methods conducted by a single operator. Comparisons by a variety of operators using the same or equivalent calibration procedures over a span of many days are necessary if the comparison results are to be credible.

Results from such multiday comparisons of KI procedures with other methods have been reported in an averaged form by several investigators. Such results have been reported in the predecessor to this criteria document[60] and more recently by Ballard et al.,[7] Stevens et al.,[100] Clark et al.,[18] and the California Air Resources Board.[16] These recent results show, in general, small differences between KI-oxidant data and ozone data. Although these differences are usually in the direction of higher oxidant than ozone, the imprecision of the calibration and operation of the KI methods makes drawing conclusions from such studies difficult. Several studies are illustrated in Figures 7-1 to 7-5.[64,69,89,91,100]

Results from side-by-side comparisons of the KI method and one O_3-specific method have been reported also for individual days and will be briefly reviewed. Davis and Jensen[21] reported measurements in Florida in which a coulometric oxidant instrument (Mast) gave readings consistently lower than those of a chemiluminescence ozone detector by a factor of three. The coulometric instruments were operated using factory calibrations, while the chemiluminescence monitors were calibrated according to specified standards. The report contains no data to demonstrate that the instrument would indicate similar ozone levels if both were sampling the same calibration atmosphere. Because of the imprecision of the calibration procedures, it is probable that a significant fraction of the discrepancy was caused by calibration. Furthermore, the differences between the two instrument readings were on the order of 20 to 30 ppb. Since no measures were taken to remove or correct for interferences from reducing agents, at least part of the difference is probably due to interference in the Mast instrument from SO_2. These uncertainties raise pertinent questions about the value of the Davis and Jensen[21] report in estimating the equivalency of the ozone and oxidant methods. EPA measurements in St. Louis, Mo., showed consistently higher KI-oxidant values relative to chemilum-O_3 values.[18] Okita and Inugami[69] reported their ozone measurements in Musashino, Japan, to be in excellent agreement with their KI-oxidant measurements (Figure 7-2). Carroll et al.,[17] using smog chamber irradiated auto exhaust mixtures, conducted and compared parallel measurements of oxidant measured by KI and of ozone measured by chemiluminescence; their comparison showed the KI values to be higher by O to 10 percent (Figure 7-3). Finally, Severs and Neal[64,89] reported the results of their comparison of the KI-oxidant and chemilum-O_3 measurements of the Houston atmosphere, which show the chemilum-O_3 values to be generally higher and, occasionally, to be considerably higher (Figures 7-4 and 7-5).

Thus with the exception of the Houston data obtained by Severs and his coworkers,[64,89] the KI-oxidant measurements appear, as a rule, to be either roughly equal to or somewhat higher than the chemilum-O_3 measurements, a difference that is directionally consistent with the difference in response specificity between these methods. The significance of the Severs results, although difficult to assess, does not seem to be crucial.

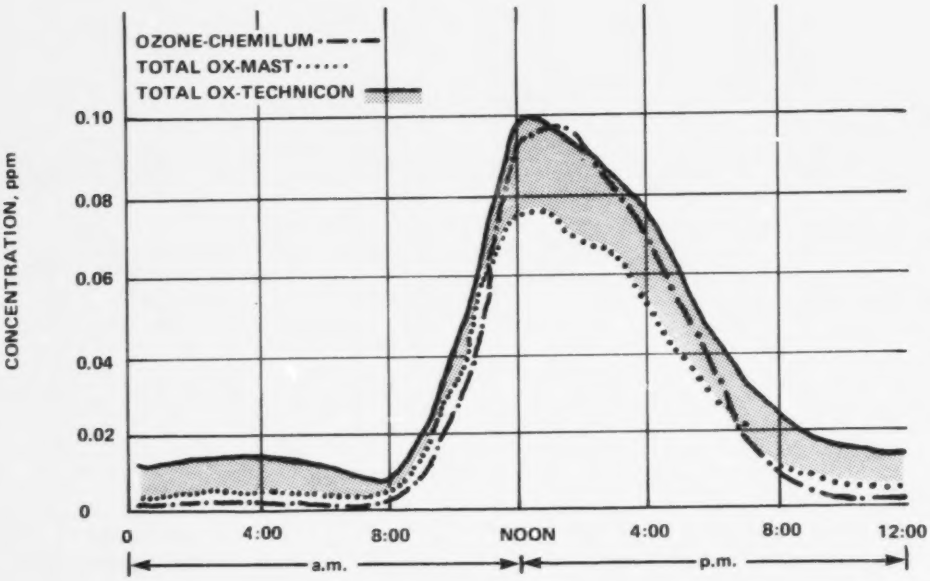

Figure 7-1. Diurnal ozone-oxidant averages from September 4 through September 30, 1971.[51]

First, it is difficult to explain the "irrational" direction of the KI-oxidant-versus-chemilum-ozone disagreement reported by Severs. Second, there are several other studies that yielded rational results. Finally, there is ample evidence attesting to the poor precision of the KI methods and, specifically, to the presence of a substantial operator error. In view of all these facts and indications, one would be inclined to conclude, presently at least, that the data analyzed by Severs reflect a strong operator error in the oxidant measurements. In a more recent study, Severs et al.[90] offer the explanation that the differences in results found in their method-comparison studies are largely the result of sampling turbulent atmospheres with instruments that use different sampling intervals. This explanation, however, is not consistent with the sustained method

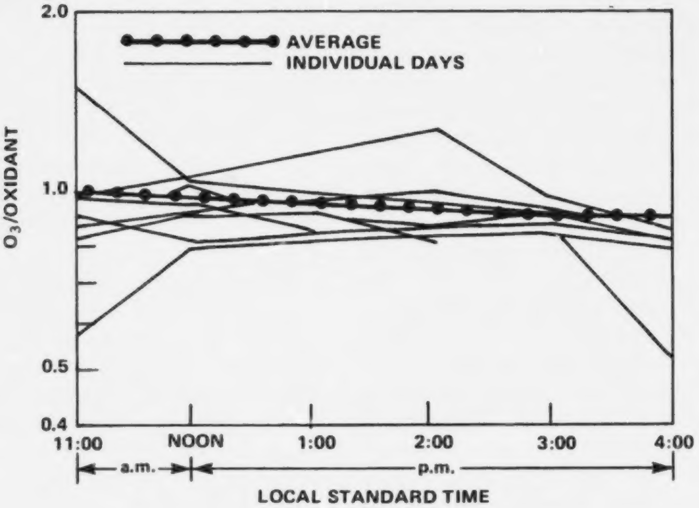

Figure 7-2. Ozone-oxidant ratios in Musashino, Japan.[69]

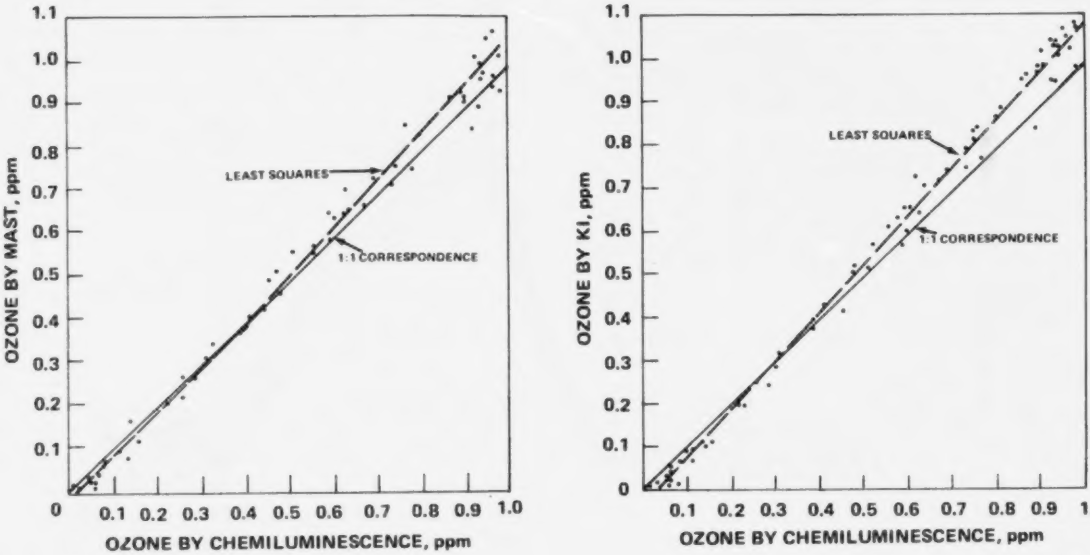

Figure 7-3. Comparison of the KI, Mast, and chemiluminescence methods for measuring ozone in irradiated exhaust mixtures.[91]

disagreement over a period of several hours, as shown in Figure 7-5.[64] The major and well-supported conclusion from the discussion in this section on the data comparison of KI oxidant versus chemilum-O_3 is that definition of air quality and of air quality standards in terms of oxidant measured by the commonly used KI methods would create problems because of the inaccuracy and imprecision of such methods. These problems would be nearly eliminated, however, if the air quality and the air standard could be defined in terms of ozone.

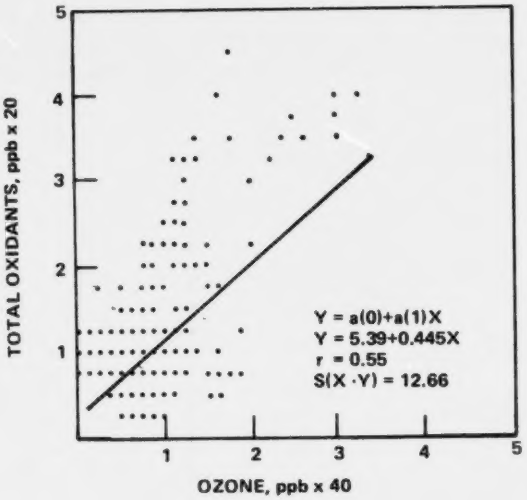

Figure 7-4. Relationship of total oxidant and ozone values greater than 10 ppb, August through December 1973.[84]

MEASUREMENT OF PEROXYACYLNITRATES

Peroxyacetylnitrate (PAN) is the principal member of a family of unstable, highly oxidized organic nitrogen compounds (peroxyacylnitrates) that are formed in polluted air by the photochemical action of sunlight on hydrocarbons and nitrogen oxides. PAN was first detected in the atmosphere and partially characterized with long-path infrared spectrometry.[87,93,95] This method, at one time, was the only means available for measuring PAN at concentrations approaching those found in polluted atmospheres. However, because of expense of the instrumentation required, the large volume of sample required, and a threshold of detectability of about 50 ppb, this method has serious limitations.

Gas chromatography with electron-capture detection evolved as a method for measuring PAN[20,44,45,94] because electron-capture detectors are very sensitive to organic nitrates and much less sensitive to hydrocarbons and simple oxygenates that would be found in atmospheric samples. Since PAN is very unstable, and because its concentration in the air is in parts per billion, the operating variables of the gas chromatographic system must be carefully chosen. PAN can be measured in polluted atmospheres at concentrations less than 10 ppb with an electron-capture detector using very small, untreated samples. Ambient air samples can be analyzed by collecting samples in a gas-sampling syringe (100 ml) and introducing this sample to the gas-sampling valve of the gas chromatograph. Samples must be analyzed at the time of collection because no reliable means of storing PAN-containing air samples is known at this time. The gas chromatographic system can be automated in such a way that samples can be taken directly from the ambient air and analyzed automatically on a cyclic, semi-continuous basis.[44]

Calibration standards for PAN cannot be purchased commercially; they must be synthesized. PAN can be synthesized at very low concentration levels by photolyzing known concentrations of ethyl nitrite vapor in an oxygen-containing atmosphere.

Continuous methods for the measurement of PAN are under development. One such method utilizes the chemiluminescence produced from the gas-phase reaction of PAN with triethylamine vapor.[74] Application of this method could result in a technique for measuring atmospheric concentrations of PAN at concentrations as low as 1 ppb.

PAN generally occurs in ambient atmospheres at concentration levels much lower than those of O_3. In addition, it is a highly reactive, unstable compound that must be treated with special precautions. For these reasons, PAN cannot be measured in a routine monitoring program. Considering the complicated methodology, the difficulty in obtaining standards, and the safety precautions required for handling PAN, only those laboratories with personnel experienced in this area should attempt to make these measurements.

Methods have been developed in which another member of the highly unstable PAN family, peroxybenzoylnitrate (PBzN), can be assayed by gas chromatography using either electron-capture[20,38,44,45,74,93,94,95] or flame-ionization detection (FID).[95] The latter method converts the

PBzN to the relatively more stable methybenzoate. The lower limit of sensitivity of this technique is 1 ppb.[5,20,38,44,45,74,94,95] With the exception of one report from the Netherlands,[5,20,38,44,45,59,74,94] the evidence indicates that PBzN does not occur in the ambient air in measurable concentrations.[5,20,38,44,45,74,94,95]

Gas chromatography with either electron-capture or FID appears presently to be the most viable method for the analyses of the PAN family.

MEASUREMENT OF HYDROCARBONS

The types of hydrocarbons covered in this discussion are those gas-phase hydrocarbons that are likely to be found in urban atmospheres. Measurement methods for these hydrocarbons fall into two classes. Those that measure nonmethane hydrocarbons (NMHC) and those that measure individual hydrocarbons.

Measurement of Nonmethane Hydrocarbons

In typical polluted air samples, the principal hydrocarbon component, methane (CH_4), is usually more abundant than all other hydrocarbons combined. Methane, however, is inert in most photochemical reactions. Therefore, hydrocarbon measurements are usually corrected for CH_4 by a subtractive technique to better approximate the reactive component of the total hydrocarbon mixtures in the ambient air. Both CH_4 and total

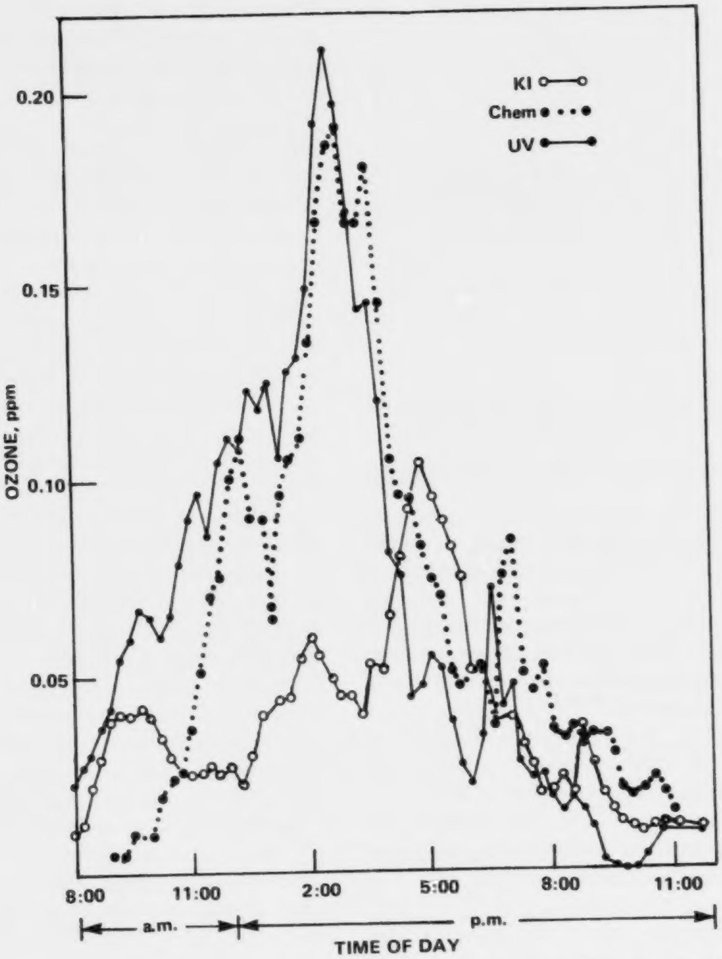

Figure 7-5. Comparative measurements of ambient oxidant/ozone by the potassium iodide (KI), chemiluminescence (Chem), and ultraviolet (UV) methods.[64]

hydrocarbons are measured by continuous monitoring analyzers that utilize the flame-ionization detector (FID) as the sensing element. Originally developed as a detector for gas chromatography, the FID was later adapted for total hydrocarbon analysis.[4] In this technique, a sensitive electrometer detects the increase in ion intensity resulting from the introduction of air containing any hydrocarbon compounds into a hydrogen flame.

The response of FID analyzers is related not only to the concentration of the hydrocarbon being measured, but also to the effective carbon number of the hydrocarbon compound. The effective carbon number varies depending on the number of carbon atoms in the molecule and on the type of compound (e.g., aliphatic, aromatic, olefinic, acetylenic, etc.). Thus, without knowing which hydrocarbon compound is being measured, the reading cannot be related to the actual concentration of the total hydrocarbons in the air sampled.

For this reason, FID results are usually expressed in terms of the calibration gas used (for example, ppm C as CH_4). Carbon atoms bound to oxygen, nitrogen, or halogens give reduced response or no response.[96] There is no response to nitrogen, carbon monoxide, carbon dioxide, or water vapor. An oxygen effect does occur, but it can be minimized by appropriate adjustment of operating condition.

The response of the FID is rapid, and with careful optimization of operating parameters and proper calibration, it is sensitive to a fraction of a ppm C as CH_4. A number of studies have been carried out on optimization of such operating variables as gas flow rates, detector temperature, and oxygen content of air, all of which can affect FID response.[11,28,31]

Early measurements of NMHC involved the use of a carbon column pretreated with CH_4.[3,70,82] This column was used to remove all other hydrocarbons from the ambient sample. A total hydrocarbon analyzer with a carbon column was operated either parallel or alternatively with a total hydrocarbon analyzer without a carbon column. Methane and total hydrocarbons were measured, and by difference, the NMHC value was computed. This method was subject to much variability because of the unpredictable behavior of carbon columns when exposed to ambient conditions. However, when the carbon column was relatively new and the NMHC concentrations were high, the method was reasonably accurate.

EPA REFERENCE METHOD

The EPA reference method for NMHC, which was promulgated in 1971, involves gas chromatographic separation of CH_4 from total hydrocarbons in an air sample followed by FID of both CH_4 and total hydrocarbons.[106] The NMHC value is obtained by subtraction of the two measurements. In a 1974 survey, some 160 users of this method were identified.[76]

Several studies have been conducted to estimate the quality of ambient air data being obtained with this method. One study[76] involved the analysis of synthetic hydrocarbon mixtures in compressed air cylinders by 16 different users of the reference method. The NMHC concentrations tested were 0.23 and 2.90 ppm. The results of this study are given in Table 7-4 and indicate the percent error from the known concentrations.

TABLE 7-4. PERCENT DIFFERENCE FROM KNOWN CONCENTRATIONS OF NONMETHANE HYDROCARBONS OBTAINED BY SIXTEEN USERS

Known concentration, ppm	% difference				
	>100	50-100	20-50	10-20	0-10
0.23	6	4	3	2	1
2.90	2	--	3	2	9

The results show that at the level of the NAAQS, 0.24 ppm carbon, most of the measurements were in error by 50 to 100 percent. At 2.90 ppm, most of the measurements were in error by only 0 to 20 percent. Thus the higher the concentration tested, the better the accuracy.

The overriding causes of the inaccuracies were the inability of the instrumentation to measure the low levels of NMHC, the complexity of the instrumentation, and the inability of the average user to identify and correct problems.

Another study[57] involved monitoring the same ambient air with five different commercial FID instruments operated by skilled professionals. The results show that different analyzer pairs agreed with 0.1 to 0.5 ppm carbon. Although these differences represent only 1 to 5 percent of the full-scale (0 to 10 ppm) range of the instruments and are normal errors for ambient monitoring, they are obviously very large relative to the NAAQS of 0.24 ppm carbon. (The 0- to 10-ppm range is necessary to include all ambient values.) Thus even under optimum operating conditions, agreement between instruments is poor at low concentrations. This result is not surprising when one realizes that the instrumentation is not optimized for measurement at these low levels.

Several studies on the performance of ambient hydrocarbon monitors have been published.[46,71,81,97] In a recent EPA-sponsored study,[35] the EPA reference method was subjected to a comprehensive evaluation, including testing of six commercial instruments. Results showed, in general, poor performance of the commercial instruments, the major problems being wide differences in response to different NMHC species and discrepancies apparently related to ambient water vapor variation.

HEAT OF COMBUSTION METHOD

A different approach to measurement of nonmethane hydrocarbons was developed in 1973.[75] This technique utilizes the fact that CH_4 has a higher heat of combustion than other hydrocarbons. One portion of the air sample passes through a catalyst bed where all hydrocarbons, except CH_4, are combusted. This CH_4-only stream passes to one FID. The other portion of the sample passes directly to a second FID for a total hydrocarbon measurement. By simultaneous processing of the signals from the two FID's and by subtraction of the CH_4-only value from the total hydrocarbon value, the NMHC value can be obtained. This technique has several advantages over the reference method. It is simpler to use, it produces continuous NMHC values, and the instrument system can maintain its own zero. However, most of the shortcomings attributed to the EPA reference method also apply to this technique.

Measurement of Individual Hydrocarbons

GAS CHROMATOGRAPHY

Gas chromatography (GC) is the most effective method for determining the concentration of individual hydrocarbons that make up the complicated hydrocarbon mixture such as is found in auto exhaust and the ambient atmosphere. With FID, the method is sensitive down to the ppb range. GC methods in general involve the separation of a complex mixture into individual compounds. This separation is achieved using both capillary (liquid phase coated on the inner surface of the column) and analytical columns. The latter may contain either a solid polymeric adsorbent (gas-solid chromatography) or an inert support coated with a liquid (gas-liquid chromatography). An inert gas (carrier gas) continuously flows through the GC column. When a complex sample is injected onto the GC column, the carrier gas carries the sample through the column. Separation of the mixture occurs as the result of the different propensities of the respective components for interacting with the column substrate. The net result is the separation of a complex mixture into a number of individual components. The degree of separation depends on column length, carrier gas flow rate, temperature, and a variety of other parameters. The column support is selected from numerous available materials, depending on the types of components that make up the sample mixture.

GC techniques have been used to investigate the hydrocarbon composition of both urban and rural atmospheres.[2,47,50,78] In urban atmospheres, hydrocarbons generally exist at levels high enough to permit the collection of samples without concentration. Samples can be collected in inert, flexible bags and transported to a central laboratory for GC analysis. Problems often arise with this sampling technique because of hydrocarbon losses resulting from adsorption on the walls of the bags, contamination of bags, and reactions that occur after the sample has been collected. Since the hydrocarbon components in rural ambient atmospheres are typically found in ppb concentrations, cryogenic trapping techniques for large air samples are required. Quantitative measurements of the GC peaks can be made with integrator and computer interfacing. Quantitative evaluation of the individual peaks can be made without standardization of each compound peak analyzed. Computation of each component concentration can be made by using an average per-carbon factor determined by known concentrations of the identified components.[25]

In recent years, the development of high-resolution capillary and support-coated, open tubular columns combined with wide-range temperature programming from subambient to elevated temperatures have provided better separation of the complex ambient hydrocarbon mixture. The complexity of the ambient air mixture, however, still prevents complete resolution of the air sample into all of its components. To determine the purity of the GC peaks and to obtain information for the identification of unknown peaks, a combination of GC with either infrared (IR) or mass spectrometry (MS) can be used. These approaches, however, require both large sample concentration and highly trained personnel.

GC - CHEMILUMINESCENCE DETECTOR

A procedure has been developed that permits detection of unsaturated hydrocarbons (e.g., ethylene, propylene, and terpenes) using separation with a gas chromatograph and analysis with a chemiluminescence detector.[56,88] The unsaturated compound of interest reacts with an ozone stream to produce light in the region of 400 Å. Chromatographic separation makes the technique specific for each compound. Sensitivity for this method is in the ppb range.[88]

INFRARED SPECTROMETRIC TECHNIQUES

Recent advances in long-path IR techniques using Fourier-transform spectroscopy (FS) have resulted in an increase in sensitivity (10 ppb) for measurement of selected hydrocarbons such as ethylene, propylene, methane, formaldehyde, and PAN. Whereas previous IR techniques required a concentration step to measure ambient levels of hydrocarbons, the FS approach allows for direct measurement at low pollutant concentrations.[33] The system has been used successfully in atmospheric studies as well as in characterization of photochemical reactions.[34] The size and complexity of the system, however, limits its usefulness for routine air monitoring.

Measurement of Reactive Hydrocarbons

Hydrocarbons differ greatly in the ability to produce photochemical smog. For example, CH_4 and acetylene can be considered unreactive, and ethylene and *n*-butane, reactive hydrocarbons. However, *n*-butane concentrations several fold higher than ethylene concentrations are required to produce similar rates of smog formation. There is accordingly a need for a measurement method that gives an indication of total hydrocarbon reactivity (i.e., a measurement that gives the sum of the concentrations of each reactive hydrocarbon species multiplied by a reactivity factor). Fontijn et al.[30] have recently developed a chemiluminescence method that appears to satisfy this need. This method is based on the *difference* between the light emission intensities at 308.9 and 312.2 nm that result from the reaction between the hydrocarbon sample and oxygen atoms under low total pressure (1 torr) conditions. A limit of sensitivity of 0.05 ppm ethylene-equivalent hydrocarbon and a linear response to individual hydrocarbons up to at least 3000 ppm have been obtained. The response to hydrocarbon mixtures appears to be additive, and interferences from CO, CO_2, SO_2, CH_4, C_2H_2 and NO_x in concentrations typical of those in auto exhaust were found to be negligible. Such an instrument response obviously measures the reactivity of the hydrocarbons with oxygen atoms. Such reactivity, in turn, is a measure—although a crude one—of the sample's ability to produce oxidants.

Calibration

Calibration at regular intervals is, of course, required for all analytical methods for measuring air pollutants. Calibration techniques and the required frequency of calibration vary for different pollutants. The preferred procedure consists of passing known concentrations of the pollutant in air into the measuring system. This dynamic procedure calibrates not only the detector response, but also the inlet system to the detector.

Calibration gases in steel or aluminum cylinders are commonly used to calibrate hydrocarbon analyzers. Cylinder gases may be diluted with clean, dry air to give the desired concentration range. Dry air with a hydrocarbon concentration that does not exceed 0.1 ppm is needed for dilution and for providing zero gas for the instrument. Calibration gases and zero gas are available commercially, and some standard gases, such as propane in air, are available from the National Bureau of Standards as standard reference materials. All commercial mixtures should be referenced against a standard gas whenever possible. The air used for instrument zeroing purposes should contain the normal constituents of clean air— that is, O_2 at 20 percent, N_2 at 79 percent, CO_2 at 320 ppm, and the normal levels of inert gases such as argon (Ar), helium (He), neon (Ne), and nitrous oxide (N_2O).

Permeation tubes can also be used to generate known concentrations of certain hydrocarbons such as butane, which can be condensed at moderate pressures. The gas trapped inside the permeation tube will diffuse through the tube wall at a rate dependent on the surface area and the temperature of the tube.[52,68] The permeation rate of the tube can be determined gravimetrically by holding the tube at a constant temperature (±0.1°C) and measuring weight loss with time. The calculated permeation rate remains constant for properly prepared tubes. Because the permeation rates of different hydrocarbons are significantly different, only one hydrocarbon can be used in a given permeation tube.

Another dynamic calibration technique involves the addition of a measured amount of pollutant to a known, fixed volume of air in a large vessel and then drawing the atmosphere into the analyzer.[66] The addition may be made by syringe injection or by the crushing of a weighted glass ampule containing the pollutant. Rigid vessels or plastic bags, which have the advantage of collapsing as the sample is withdrawn, may also be used. Bags must be made of an inert plastic to avoid changes in concentration through absorption or reaction with the walls of the bag. The bags must also be checked for permeation rates of the compounds being used, since permeation can result in loss of sample or, in some cases, contamination of the sample as a result of bag permeation by ambient hydrocarbons. The above techniques are preferred for more reactive hydrocarbons that do not have sufficient stability to be stored in cylinders.

MEASUREMENT OF NITROGEN OXIDES

The NO_x involved in photochemical oxidant formation are NO and NO_2. Since oxidant control regulations and practices to date have been based on unilateral control of the hydrocarbon precursor, interest in the NO_x factor was only peripheral and of research nature. The recent findings, however, on the need to consider the NO_x factor in formulating oxidant control strategies have raised questions regarding adequacy of the existing analytical methods for ambient NO_x measurement. Since the primary needs insofar as the oxidant problem is concerned are short-term (hourly) NO_x concentration data for urban atmospheres and short-term (hourly) data for rural atmospheres, analytical methods used for oxidant-related applications must be capable of short-term sampling and analysis and interference-free response, and they must be sensitive. This discussion will thus be limited to continuous methods for measuring NO_2 and NO.

Measurement of NO_2

CHEMILUMINESCENCE METHODS (EPA REFERENCE METHODS)

On December 1, 1976, EPA promulgated a new measurement principle and calibration procedures for reference methods for measuring NO_2.[105] The measurement principle and calibration procedures are described in Appendix F of 40 CFR Part 50. The measurement principle is based on the gas-phase reaction of O_3 and NO. Atmospheric concentrations of NO_2 are measured indirectly by photometrically measuring the light intensity (at wavelengths greater than 600 nm), resulting from the chemiluminescent reaction of NO with O_3.[29,54,99] NO_2 is first quantitatively reduced to NO[13,42,99] by means of a converter. NO, which commonly exists in ambient air together with NO_2, passes through the converter unchanged, causing a resulting total NO_x concentration equal to NO + NO_2. A sample of the input air is also measured without having passed through the converter. This latter NO measurement is subtracted from the former measurement (NO + NO_2) to yield the final NO_2 measurement. The NO and NO + NO_2 measurements may be made concurrently with dual systems or cyclically with the same system. Two calibration procedures are prescribed in Appendix F of 40 CFR Part 50. One is a gas-phase titration procedure referenced to an NO-in-nitrogen standard. The other calibration procedure is referenced to an NO permeation device. Both of these standards can be obtained from the National Bureau of Standards as standard reference materials. Chemiluminescence $NO/NO_x/NO_2$ analyzers will respond to other nitrogen-containing compounds such as PAN, which might be reduced to NO in the thermal converter.[107] Atmospheric concentrations of these potential interferences are generally low relative to NO_2, and valid NO_2 measurements may be obtained.

GRIESS-SALTZMAN METHODS

Before the development of chemiluminescence analyzers, most NO_2 data were collected using methods based on variations of the Griess-Saltzman[83] methodology. These methods are based on the specific reaction of the nitrite ion (NO_2^-) with diazotizing-coupling reagents to form a deeply colored azo dye. The NO_2 in the ambient air is converted to nitrite ion on contact with an absorbing solution. As applied to continuous analyzers,[84] the absorbing solution contains the diazotizing-coupling reagents, and the absorbance of the azo dye is measured continuously with a flowing photometer cell. The absorbance of the azo dye solution is directly proportional to the concentration of NO_2 absorbed.

For ordinary atmospheres, interferences have been claimed to be of negligible importance. The method is commonly calibrated with standard nitrite solutions, assuming 0.72 mole of nitrite to be equivalent to 1 mole of NO_2. This latter assumption has been at issue.[86] In addition, there are indications of an ozone interference problem.[8] For all practical purposes, this method has been replaced with the chemiluminescence method.

Measurement of NO

Before the advent of the chemiluminescence method, NO measurements were made almost exclusively by contacting the air sample with a solid or liquid oxidant and measuring resulting NO_2 by conventional azo dye colorimetric procedures.[48,49] Currently, the chemiluminescence method is believed to be unquestionably superior to all methods available. Thus, for example, relative to the continuous Griess-Saltzman, the chemiluminescence method is more accurate because it can be calibrated more accurately with standard NO mixtures, and also because it is subject to practically no interferences from other vapors. The speed of response of chemiluminescence-NO instruments is typically only a few seconds, and their sensitivity is about 5 ppb. Overall, such instruments are practical, reliable, and suitable for field use.

SUMMARY

The chemiluminescence method, based on the gas-phase reaction of O_3 with ethylene, was adopted by EPA as the reference method and is now being used extensively. Several techniques using commercial analyzers have been designated as reference methods under the EPA ambient air monitoring reference and equivalent methods regulations. Data indicate that the performance of these analyzers is better than that specified by the EPA regulations. Continuous analyzers based on gas-solid chemiluminescence and UV photometry have been developed, and techniques using such analyzers have been designated as equivalent methods.

The methods for measuring total oxidants are based on the oxidation of KI and the electrochemical or colorimetric detection of I_2. Nitrogen dioxide causes a positive interference, and SO_2 results in negative interference. Serious problems arise, however, with methods for correcting for SO_2 and NO_2 interferences.

Extensive studies in recent years have shown the various KI calibration procedures for oxidant or O_3 analyzers to be deficient. These methods suffer from systematic, accuracy-related errors as well as from random, precision-related errors. EPA intends to study and evaluate several new calibration procedures and to amend Appendix D of 40 CFR Part 50 to revise the 1-percent NBKI calibration procedure or replace it with one or more of several alternatives under consideration. The alternative procedures under consideration are: (1) GPT with excess NO; (2) UV photometry; (3) GPT with excess O_3; and (4) boric acid KI.

Analysis of the data indicates that oxidant-by-KI measurements of oxidant levels are generally comparable or slightly higher than levels of ozone as measured by ozone-specific techniques. However, most oxidant/O_3 comparison studies suffer from the imprecision of the oxidant methods and from the lack of data regarding levels of compounds known to interfere with the KI method.

Commercial instruments for ambient total NMHC monitoring have been tested extensively and found to be inaccurate at levels approaching the 0.24-ppm C air quality standard. At present, ambient hydrocarbons can be accurately measured only by sophisticated gas chromatography. Routine methods are, however, under development.

On December 1, 1976, EPA promulgated a new measurement principle and calibration procedure for measuring NO_2. The measurement principle is based on the gas-phase chemiluminescent reaction of O_3 and NO. Two calibration procedures are prescribed. One is a GPT procedure referenced to an NO-in-nitrogen standard. The other procedure is referenced to an NO_2 permeation device. Before the development of chemiluminescence analyses, most NO_2 data were collected using methods based on variations of the Griess-Saltzman methodology. For all practical purposes, this method has been replaced with the chemiluminescence method.

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8. TOXICOLOGICAL APPRAISAL OF PHOTOCHEMICAL OXIDANTS

INTRODUCTION

Photochemical oxidants such as ozone and peroxyacylnitrates are gases that exert their toxic effects by entering the body through inhalation. If present in sufficient concentrations, they are capable of causing death to various organisms. At sublethal concentrations, they may alter, impair, or otherwise interfere with physiological processes.

Alterations in pulmonary function and in mechanical properties of the lungs are among the effects that result from inhalation of these compounds. Other effects include morphological changes in the lungs, increased susceptibility to infectious respiratory disease, and biochemical alterations in the lungs and in other organs. Since there is a scientific consensus that concentrations above 1960 $\mu g/m^3$ (1 ppm) ozone are very hazardous to health, generally only research conducted at or below this concentration will be described here.

The toxicological effects of ozone and two major pollutant groups, oxidants (mixtures of substances produced by photochemical reactions) and peroxyacylnitrates, are discussed in this chapter. Each compound or group is treated separately.

EFFECTS OF OZONE ON EXPERIMENTAL ANIMALS

Respiratory Tract Transport and Absorption

NASOPHARYNGEAL UPTAKE

The extent to which air pollutants can directly affect the lung depends largely on the amounts of pollutant and reaction products that penetrate to the lower airways. Nasopharyngeal removal of ozone lessens the insult to the lung and must be accounted for when estimating ozone concentrations reponsible for observed pulmonary effects. Experimental estimates of nasopharyngeal ozone removal determine the appropriate boundary conditions when using convective/diffusion equations to model ozone transport in the lower airways.

Vaughn et al.[200] exposed the isolated upper airways of beagle dogs to ozone at a continuous flow of 3.9 l/min and collected the gas just below the larynx in a plastic (Mylar) bag. Concentrations below 784 $\mu g/m^3$ (0.4 ppm) yielded essentially 100-percent uptake. However, Yokoyama and Frank[215] observed 60- to 70- percent uptake of ozone by the upper airways in similar studies on beagles where Teflon tubing was used. When these investigators repeated the procedure of Vaughn et al.,[200] they found that some ozone was lost because of adsorption on the bag wall.

Nasal uptake significantly exceeded ($p<0.01$) oral uptake at both flow rates (3.5 and 35 l/min) used in the Yokoyama and Frank[215] studies. The overall uptake coefficient for the nose increases with air flow rate. This latter point was shown by Aharonson et al.,[1] who demonstrated the necessity for expressing retention data on the basis of the average uptake coefficient when comparing uptake at different flow rates.

The decomposition of ozone in the nasopharynx of acutely versus chronically exposed beagle dogs was studied by Moorman et al.[139] Dogs chronically exposed (18 months) to 1960 to 5880 $\mu g/m^3$ (1 to 3 ppm) ozone, using various daily exposure regimens, were shown to have significantly higher mean tracheal concentrations of ozone than animals tested after 1 day of exposure to the corresponding regimens. These investigators postulated that the differences were likely to be due to physiochemical alterations of the mucosal lining in the chronically exposed animals.

When dogs were chronically exposed to 1960 $\mu g/m^3$ (1 ppm), those exposed for 8 hr/day had significantly lower tracheal values than those continuously exposed. However, the average concentration for the acute exposure was not different from that of the 8 hr/day chronic exposure group (19.6 vs 44.1 $\mu g/m^3$, 0.01 vs 0.023

ppm). If one takes into account the sensitivity of the Mast ozone meter used to measure the responses, this latter difference would not represent a significant difference in treatment unless the Mast ozone meter had been modified. Moorman et al.[139] did not state what modifications, if any, were performed. Nevertheless, in view of the small difference observed with exposure to 1960 $\mu g/m^3$ (1.0 ppm), it is likely that chronic exposure to lower levels would not result in tracheal ozone concentrations significantly greater than those observed with acute exposure.

Nasopharyngeal removal of ozone in rabbits of both sexes and in male guinea pigs has been studied by Miller[134] over a concentration range of 196 to 3920 $\mu g/m^3$ (0.1 to 2.0 ppm). The tracheal ozone concentration in these species was markedly similar and was linearly related to the chamber concentration of ozone drawn undirectionally through the isolated upper airways. Regression analyses showed that ozone removal in the nasopharyngeal region is approximately 50 percent in both species. No difference in ozone removal was observed between male and female rabbits.

TRACHEOBRONCHIAL UPTAKE

Lower airway removal of ozone from inspired air was measured by Yokoyama and Frank[215] in dogs that were mechanically ventilated through a tracheal cannula. Two ranges of ozone concentration were studied: (1) 1372 to 1666 $\mu g/m^3$ (0.7 to 0.85 ppm) and (2) 392 to 784 $\mu g/m^3$ (0.2 to 0.4 ppm). The rate of uptake was found to vary between 80 and 87 percent when the tidal volume was kept constant and the respiratory pump was operated at 20 to 30 cycles/min. This estimate of uptake applies to the lung as a whole and does not indicate the uptake of ozone by individual airway generations.

A major goal of environmental toxicological studies on animals involves the eventual extrapolation of the results to man. In addressing the likelihood that the animals studied mimic the human response, estimation of the effective dose delivered to the target organ is required. Also, differences between man and experimental animals in the ratio of effective dose to exposure concentration must be considered. Extensive lower airway morphometric data on human beings,[206] guinea pigs, rabbits, and rats,[111] and the technical inability of obtaining local lower airway ozone uptake data make mathematical modeling the method of choice for examining ozone uptake in the deep lung.

The mathematical model of lower airway ozone uptake by McJilton et al.,[125] although widely cited, has never been formally published. However, the major features of the McJilton et al. model have been described in some detail, and model results for absorption of ozone in each generation of the human tracheobronchial tree have been presented.[155] Since nonreactivity of ozone with the mucous layer was assumed, the model of McJilton et al. is really more useful for estimating lower airway uptake of water soluble and relatively water insoluble gases that are nonreactive with the mucous layer than it is for estimating the uptake of ozone.

The problem of treating chemical reactions of ozone with various components of mucus has been included in the mathematical model of transport and removal of ozone developed by Miller.[134] These reactions were shown to be characterized by an instantaneous reaction regime. Three cases, which are a function of the lumen ozone concentration, were used to characterize the removal of ozone from an airway. As did the model of McJilton et al.,[125] Miller included the effects on gas transport of convection, axial diffusion, and radial diffusion, although the distance-time grid mesh used was 5-fold smaller than that used by McJilton et al. Also, Miller treated the effects of both molecular diffusion and eddy diffusivity on axial diffusion, rather than including only molecular diffusion.

Miller et al.[136] modeled tracheobronchial uptake of ozone in the lungs of guinea pigs and rabbits, as well as man. The predicted pulmonary ozone dose curves obtained indicate that a general similarity exists between these species in the shape of the dose curves. Independent of the inhaled tracheal concentration of ozone, the respiratory bronchioles are predicted to receive the maximum dose of ozone, a result that is in good agreement with experimental findings in various animal species.[50,190,191]

The model predicts uptake of ozone by respiratory airway tissue for all tracheal concentrations studied (62.5 to 4000 $\mu g/m^3$, or 0.03 to 2.04 ppm). However, penetration of ozone to the tissue in airways lined by mucus is not predicted for inhaled tracheal concentrations less than 125 $\mu g/m^3$ (0.06 ppm) in man and 62.5 $\mu g/m^3$ (0.03 ppm) in guinea pigs and rabbits. As the inhaled tracheal concentration is increased from these low levels, the tissue deposition pattern of ozone in the conducting airways becomes smoother, increases in magnitude, and includes more airways. Alveolar ozone doses decline sharply from the maximum values predicted for the respiratory bronchioles.

As the tracheal ozone concentration decreases from approximately 100 $\mu g/m^3$ (0.05 ppm), there is a more gradual decline in the respiratory bronchiolar dose curve for guinea pigs and rabbits than there is in the curve for man. However, for any given tracheal concentration greater than 100 $\mu g/m^3$ (0.05 ppm), the predicted respiratory bronchiolar dose for rabbits is 80 percent of that for man and twice that for guinea pigs. With tidal volumes corresponding to mild exercise, a 2.4-fold increase in ozone dose to the respiratory bronchioles of man is predicted. This dosage increase correlates with pulmonary function effects observed with exposure of human subjects to ozone while exercising.[10]

Mortality

Mittler et al.[137] have determined the LD_{50} for ozone in a variety of laboratory animals. The LD_{50} of a toxic substance is defined as that dose expected to kill 50 percent of a population of experimental animals. For common laboratory animals such as mice, rats, guinea pigs, and cats, 3-hr exposures of $\geq 41{,}160\ \mu g/m^3$ (21 ppm) are required to reach the LD_{50}. Mice treated with selected antioxidants can be partially protected against mortality from high dose ozone exposures.[132,159,174,175] Concentration and length of exposure are not the only factors that determine toxicity. Stokinger cited other factors that influence the response to a specific level of ozone.[194] These include age, temperature, exercise, dosage rate, respiratory infection, and reducing agents.

In addition to these factors, Skillen[189] confirmed the observation of Fairchild et al.[63] of a variation in the effect of continuous ozone exposure to 11,800 $\mu g/m^3$ (6 ppm) on rats with varying thyroid status. The average survival times of these rats were as follows: More than 10 hr for those rats with reduced thyroid function, 6.7 hr for rats with unaltered thyroid activity, and 2.2 hr for rats with stimulated thyroid function.

Pulmonary Effects

HOST DEFENSE MECHANISMS

The adverse effects of a number of air pollutants, including ozone, on pulmonary defense mechanisms against infectious disease have been studied. This section will describe how low concentrations of ozone result in an enhancement of mortality in animals exposed to bacterial aerosols. This increased susceptibilty to pneumonia is ascribed primarily to dysfunction of the primary defense cells of the lungs, the alveolar macrophages.

Interaction with Infectious Agents — The effects of a number of pollutants on a host challenged with infectious microorganisms have been reviewed.[36,77] Briefly, mice are exposed either to ozone or filtered clean air. At various time periods, all of the animals are challenged with an aerosol of viable microorganisms, and the accumulated mortality over a 15-day observation period in clean air is reported. The concentrations of pollutants used alone (i.e., without infectious agents) caused no deaths.

Coffin et al.[35] exposed mice to different concentrations of ozone ranging from 137 to 981 $\mu g/m^3$ (0.07 to 0.5 ppm). The 3-hr exposures were followed immediately by bacterial aerosol exposure (*Streptococcus pyogenes*, Group C). With the exception of the lowest ozone concentration, 137 $\mu g/m^3$ (0.07 ppm), each concentration was tested once (40 mice/concentration). It was reported that $\geq 157\ \mu g/m^3$ (0.08 ppm) ozone resulted in a significant ($p < 0.05$) enhancement of mortality of at least 23 percent. As the concentration of ozone was increased up to 1019 $\mu g/m^3$ (0.52 ppm), there was a trend toward increased mortality.

Ehrlich et al.[54] conducted studies using the same microorganism as Coffin et al.,[35] but with different strains of mice. Gas concentrations were monitored by the chemiluminescence method. A single 3-hr exposure to 196 $\mu g/m^3$ (0.1 ppm) ozone caused a significant ($p < 0.05$) increase in mortality.

Using CD-1 mice and *S. pyogenes*, Miller et al.[135] compared the effects of a 3-hr exposure to ozone at 196 $\mu g/m^3$ (0.1 ppm) in which the bacterial aerosol was administered either immediately, 2, 4, or 6 hr after cessation of the ozone exposure. In each replicate experiment, 20 mice per group were used, and ozone was monitored continuously by chemiluminescence. For these post-exposure challenges, only the 2 hr time resulted in a significant ($p < 0.05$) increase in mortality (6.7 percent) over controls. However, when the animals were infected with *Streptococci* during the ozone exposure, a significant ($p < 0.01$) increase in mortality of 21 percent was observed. When this latter experimental regimen was used, exposure to 157 μg/m^3 (0.08 ppm) ozone resulted in a significant ($p < 0.05$) increase in mortality of 5.4 percent.

The differences in results among these studies may have been due to a variation in the sensitivity of method of ozone monitoring, a difference in mouse strain, changes in the pathogenicity of the bacteria, or differences in sample size.

The interactions of other environmental stresses (cold and other pollutants) using the infectivity model have also been examined. Using *S. pyogenes* as the infectious agent, Coffin and Blommer[34] showed that animals preexposed to cold (6° to 9°C, or 43° to 48°F) for 3 hr had a greater mortality after a 3-hr exposure to 1372 to 1764 μg/m^3 (0.7 to 0.9 ppm) than did animals exposed to ozone only.

The effects of exposures to mixtures of NO_2 and ozone were examined by Ehrlich et al.[54] using the infectivity model. Significant ($p < 0.05$) increases in mortality occurred when mice were exposed to concentrations at or above 3926μg/m^3 (2 ppm) NO_2 and 98 μg/m^3 (0.05 ppm) ozone for 3 hr before challenges with aerosols of *Streptococcus*. Although not statistically tested, the effect of the exposure to the combination appeared to be additive (i.e., the increases in mortality were approximately equivalent to the sum of those induced by the exposure to each individual pollutant. In addition, the animals exposed to the mixture show a reduced rate of bacterial clearance from the lung.

Gardner et al.[80] examined the effects of sequential exposure to 196 μg/m^3 (0.1 ppm) ozone for 3 hr and 0.9 mg sulfuric acid (H_2SO_4)/m^3 (0.3 μm) for 2 hr using CD-1 mice. The bacterial aerosol was administered at the end of the pollutant exposure. When the ozone preceeded the H_2SO_4 exposure, there was a significant increase in mortality ($p < 0.05$), which was additive.

The results derived from studies utilizing the infectivity model indicate its sensitivity for detecting biological effects at low pollutant concentrations and its response to modifications in technique (i.e., using different mouse strains or varying the time of bacterial challenge). The model is supported by experimental evidence (discussed in the following section and reviewed in reference 24) that shows that pollutants, albeit at different concentrations, that cause an enhancement of mortality in the infectivity system also cause reductions in essential host defense systems, such as pulmonary bactericidal capability, the functioning of the alveolar macrophage, and the cytological and biochemical integrity of the alveolar macrophage.

Coffin and Blommer[34] examined the mechanism of action of the infection following pollutant exposure. In these studies, the number of bacteria in the lung and the presence of bacteria in the blood was followed at various time periods. Mice were exposed to 1960 μg/m^3 (1 ppm) ozone for 3 hr before a bacterial aerosol. Bacterial invasion of the blood began 2 days after ozone exposure. Additional research[90] has shown that exposure to ozone concentrations greater than 780 μg/m^3 (0.4 ppm) results in lower deposition rates of inhaled *Streptococcus*. Nevertheless, the bacteria that are deposited multiply, so that shortly after exposure, there are more bacteria in the ozone-exposed lungs than in the lungs of control animals. Again, it was hypothesized that this oxidant gas adversely affected pulmonary defense systems other than mucociliary clearance.

Goldstein et al.[73,89] exposed mice to an aerosol of ^{32}P-labeled *Staphylococcus aureus* either after a 17-hr exposure to ozone or before a 4-hr exposure. Concentrations of ozone were 1180, 1370, 1570, or 1960 μg/m^3 (0.6, 0.7, 0.8, or 1 ppm), or higher. The mechanical clearance and bactericidal capabilities of the lung were then measured 4 to 5 hr after bacterial exposure. Exposure for 17 hr before infection caused a significant ($p < 0.05$) reduction in bactericidal activity beginning at 1960 μg/m^3 (1 ppm). When mice were exposed to ozone for 4 hr after being infected, there was a significant decrease in bactericidal activity for each ozone exposure level. The lowest level of measurable effect was 1180 μg/m^3 (0.6 ppm). With increasing ozone concentration, there was a progressive decrease in bactericidal activity. The investigators proposed that because mucociliary clearance was unaffected by subsequent ozone exposure, the bactericidal effect was due to dysfunction of another type of pulmonary defense, most probably the alveolar macrophage. Other workers[203] found that exposure to 980 μg/m^3 (0.5 ppm) of ozone, 16 hr/day for 7 months had no effect on the mechanical clearance of polystyrene and iron particles from rabbit lungs. In the guinea pigs exposed to the same concentration for 16 hr/day for 2 months, there was only a small reduction in bacterial clearance.

Warshauer et al.[203] demonstrated that a deficiency of vitamin E augmented the adverse effect of ozone at 1374 μg/m³ (0.7 ppm) on murine pulmonary bactericidal capacity only after a prolonged exposure (7 days). Identical hypovitaminosis failed to influence pulmonary bactericidal activity in rats exposed for only 4 hr to 980 to 1960 μg/m³ (0.5 to 1.0 ppm) of ozone.

The effect of pollutant combinations on the bactericidal function of the lung has also been examined.[84] Some mice received an intratracheal injection of 10 mg of silica, and others received latex. Seventy days later, the animals were infected with ^{32}P-labeled *Staphylococcus aureus* and exposed to either 4 hr of 785 μg/m³ (0.4 ppm) ozone or to clean air. Both the silicotic and latex-injected (control) mice had similarly reduced levels (12 percent) of bactericidal activity after breathing ozone ($p < 0.05$), when compared to their ozone-exposed counterparts. Silicosis itself did not inhibit the pulmonary bactericidal response. Goldstein et al.[91] conducted a similar study in which mice were exposed to ozone/NO_2 combinations: From 196 to 790 μg/m³ (0.1 to 0.4 ppm) ozone, and from 3760 to 13,720 μg/m³ (2 to 7.3 ppm) NO_2 either for 17 hr before infection or for 4 hr after infection. The lungs were capable of functioning well until the higher pollutant level was reached. Physical clearance was not affected at either level. The demonstrated injuries were those that would be expected from each individual oxidant.

Fairchild[62] found that mice exposed for 1 hr to 1770 μg/m³ (0.9 ppm) ozone exhibited a 70-percent increase in respiratory deposition of vesicular stomatitis virus ($p < 0.005$) occurring in the nasal cavity rather than in the lung. Also, the minute ventilation was reduced ($p < 0.05$) by a maximum of 30 percent at the end of the exposure. Fifteen minutes later, the minute volume increased but remained lower than that of the controls.

An ozone concentration of 1177 μg/m³ (0.6 ppm) for 3 hr caused an inhibition of replication of influenza virus (A2/Japan, 305/57) deposited in the nasal cavities of mice. In contrast, a slight increase in growth of vesicular stomatitis virus was seen in the noses of mice exposed to 1767 μg/m³ (0.9 ppm) ozone for 3 hr.[61]

Laboratory-induced parasitic infection (*Plasmodium berghei*) of mice was also exacerbated by exposure to 1686 μg/m³ (0.86 ppm) ozone for 8 hr/day, 5 days/week for 6 months.[182]

Alveolar Macrophages — In most of the foregoing studies, the authors recognized the possibility that damage to the alveolar macrophages (AM) was responsible primarily for the enhanced infectivity and reduced bactericidal response. Recent investigations have further advanced this hypothesis. When rabbits were exposed to 980 μg/m³ (0.5 ppm) or 1313 μg/m³ (0.67 ppm) for 3 hr, there was a significant reduction ($p < 0.001$) in the number of bacteria phagocytized by AM.[36,38] Because phagocytosis begins with particle attachment to the cell membrane, increased fragility of the AM could play a role. Rabbits exposed to 980 μg/m³ (0.5 ppm) for 8 hr/day for 7 days exhibited a trend toward increased membrane fragility of AM, although lipid peroxidation was undetectable at this level.[48]

The AM within the lung reside within a liquid layer. Gardner[76] demonstrated that the protective components of this layer are inactivated by a 2.5-hr exposure to 196 μg/m³ (0.1 ppm) ozone. When normal or ozone-exposed AM were placed in fluid lavaged from exposed rabbits, they showed more lysis (10 percent over controls). A similar effect was seen when normal AM were placed in the protective fluid that had been exposed in vitro to ozone at 196 μg/m³ (0.1 ppm) or 1960 μg/m³ (1 ppm) for 30 min.

Goldstein et al.[85] studied the effect of a 2-hr ozone exposure on the ability of AM to be agglutinated by concanavalin A, a parameter reflecting membrane organization. A decrease in agglutination of rat AM was found after exposure to 980 or 1960 μg/m³ (0.5 or 1.0 ppm). A decrease in concanavalin A agglutinability of trypsinized red blood cells obtained from rats exposed for 2 hr to 1960 μg/m³ (1 ppm) was also noted. Hadley et al.[94] investigated AM membrane receptors from rabbits exposed to 980 μg/m³ (0.5 ppm) ozone for 3 hr. Following ozone exposure, lectin treated AM have increased rosette formation ($p < 0.05$) with rabbit red blood cells. The authors hypothesized that the ozone-induced response indicates alterations of macrophage membrane receptors for the wheat germ agglutinin that may lead to changes in the recognitive ability of the cell.

Hurst et al.[106] showed that the acid hydrolases of AM lysosomes are significantly reduced after rabbits are exposed for 3 hr to either 490 μg/m³ (0.25 ppm) or 980 μg/m³ (0.5 ppm) of ozone. Greatest reductions (25 percent and 40 percent, respectively) were seen in the lysozyme activity. Acid phosphatase and β-glucuronidase activities were also affected. Since these enzymes are involved in intracellular degradation of bacteria, their reduced activities could contribute to poor macrophage functioning and, consequently, to enhanced mortality from bacterial invasion. Similar enzymatic reductions were observed in AM exposed in tissue culture.[105] The effects of ozone on host defenses were also studied by means of unilateral lung exposures of rabbits.[3] Three hours of ozone exposure was found to decrease cellular viability, depress various intracellular enzymes, and increase the number of pulmonary polymorphonuclear leukocytes. The effects were dose-related, beginning at 980 $\mu g/m^3$ (0.5 ppm). The responses were found to be specific to the lung that breathed ozone rather than any generalized systemic response.

In vitro techniques have also been employed to study AM function and biochemistry after ozone exposure. Weissbecker et al.[207] established dose-response curves for the effect of ozone on the viability of AM, with effects being seen at 118 $\mu g/m^3$ (0.06 ppm). Richmond[173] studied the in vitro phagocytic activity of AM using radioisotope-labeled bacteria. Exposure of adhered AM to ambient air or to 1570 $\mu g/m^3$ (0.8 ppm) ozone resulted in little difference in uptake of ^{32}P by *Staphylococcus albus*. The exposed AM engulfed fewer bacteria than controls. The concentration of ozone was not measured either during or following the 2-hr exposure.

Evidence that ozone may be inhibitory to interferon (an antiviral substance) synthesis was studied by Ibrahim et al.[123] Mice were exposed for 14 or 21 days to 1570 $\mu g/m^3$ (0.8 ppm) ozone. At the end of the exposure periods, some mice were removed to ambient air for recovery studies. Tracheal explants and AM from the exposed mice were cultured. The data indicate that for the first 10 days of ozone exposure, no effect was observed on the capacity of the tracheal epithelial cells to produce interferon. However, starting on the 11th day, there was a continual decrease in interferon production. In the recovery studies, it was found that 10 days after the mice were moved to clean air, the epithelial cells began to produce a small amount of interferon. Complete recovery occurred 24 days after removal to clean air. The authors reason that the results could be due to a decreased synthesis of interferon or an inhibition of interferon release from the tissues. No effect on the capacity of AM to produce interferon was seen after 21 days of exposure.

Immunology — Matsumura[123] exposed albumin-sensitized guinea pigs to 1960 $\mu g/m^3$ (1.0 ppm) ozone and found no increases in anaphylactic attacks after subsequent antigen inhalation. In a further study, Matsumura et al.[124] found that guinea pigs that received a 1-hr pretreatment with 1960 $\mu g/m^3$ (1 ppm) ozone had no anaphylactic responses to inhaled acetylcholine, a bronchoconstrictor agent, after a later exposure to 3930 $\mu g/m^3$ (2.0 ppm) ozone.

In an attempt to define a mechanism for the edemagenic effect of ozone, Easton and Murphy[52] noted that ozone-exposed guinea pigs appeared to be more susceptible to the toxic action of histamine, a vasoactive substance released by certain cells in allergic reactions. It was observed that pre-exposure to 9800 $\mu g/m^3$ (5.0 ppm) ozone for 2 hr followed by a histamine challenge of 0.9 to 1.4 mg/kg body weight (injected 1.5 to 2.0 hr after the end of exposure) resulted in increased mortality compared with an air-exposed control group. Autopsy of the dead animals indicated that members of both groups died of acute bronchoconstriction. The increased susceptibility to histamine was detectable for 12 hr after the termination of exposure to ozone. The minimum concentration of ozone that produced an increase in mortality was 980 to 1960 $\mu g/m^3$ (0.5 to 1.0 ppm), one-twentieth of that required to produce death from pulmonary edema resulting from ozone alone. Since there was no significant difference in lung water content between the two groups, it was concluded that pulmonary edema did not contribute to the increased susceptibility. Increased mortality was found to occur only when exposure to ozone took place before challenge with histamine and not when guinea pigs were injected with histamine before exposure.

BIOCHEMISTRY

The actual mechanism of the toxicity of ozone at the subcellular level is unclear. The following theories have been advanced about the biochemical damage caused by ozone: (a) Nonspecific stress with the release of histamine, (b) oxidation of sulfhydryl groups or their precursors, (c) oxidation of polyunsaturated lipids contained mainly in cell membranes, (d) formation of toxic compounds (ozonides) through reaction with polyunsaturated lipid, (e) formation of free radicals, and (f) injury mediated by a pharmacologic action (i.e., via a neurohormonal mechanism). These mechanisms have been discussed in several reviews.[39,129,130,154,188]

Amino Acids and Proteins — The susceptibility of aqueous solutions of amino acids to oxidation in vitro from ozone exposure was ranked by Mudd et al.[140] From high to low susceptibility, the ranking is as follows: Cysteine, methionine, tryptophan, tyrosine, histidine, cystine, and phenylalanine. Other amino acids were unaffected. Sulfhydryl compounds were found to the be most susceptible to oxidation. In addition, when avidin was exposed to ozone, its ability to bind to biotin was lost, presumably because of oxidation of tryptophan residues. In vitro ozone exposure [169] to formic acid solutions of amino acids was found to have the following decreasing order of reactivity: Tryptophan, methionine, cystine, tyrosine. Other amino acids were not tested.

Protein synthesis of rat lung tissue is altered by continuous exposure to 1570 μg/m^3 (0.8 ppm) ozone, as described by Mustafa et al.[149] After 1 day of exposure, there was no difference in the in vitro incorporation of labeled amino acids into the lung slices. However, 35- and 84-percent increases in incorporation were observed after 2 and 3 days of exposure, respectively. Throughout the remainder of the study (up to 7 days), the level remained elevated and unchanged. When in vivo amino acid incorporation procedures were used, there was a 50-percent decrease in incorporation after 1 day of exposure. On days 2 and 3, there were increases of 60 percent and 100 percent over control. A plateau was reached on day 3 that extended to day 7. Under the in vivo conditions, no radioactive incorporation of amino acids occurred in the AM obtainable by lavage. Similar studies conducted in the presence of puromycin (an inhibitor of protein synthesis) showed that the increase in amino acid incorporation observed in vitro and in vivo for the ozone-exposed animals was reflected in an increase in protein synthesis.

The influence of ozone on lung prolyl hydroxylase (an enzyme thought to be rate-limiting in collagen synthesis) and the product of its reaction, hydroxyproline, was studied by Hussain et al.[108] Rats were exposed continuously to either 390, 980, or 1570 μg/m^3 (0.2, 0.5, or 0.8 ppm) ozone. At the end of a 7-day exposure to the lowest ozone concentration, no significant changes in prolyl hydroxylase activity were found. However, by 7 days there were statistically significant increases of 150 and 200 percent in the enzyme activity of lungs from animals exposed to 980 or 1570 μg/m^3 (0.5 or 0.8 ppm) ozone, respectively. For the 980 μg/m^3 (0.5 ppm) ozone treatment group, the increased activity of prolyl hydroxylase had partially returned to normal by 30 days, but it was still elevated ($p < 0.05$) over the controls. After 60 days of exposure to 980 μg/m^3 (0.5 ppm) ozone, this enzyme activity was not significantly different from control. When prolyl hydroxylase activity and hydroxyproline levels were determined in the groups receiving 1570 μg/m^3 (0.8 ppm) for 7 days, it was found that enzyme activity was elevated ($p < 0.05$) beginning at day 1. Hydroxyproline levels had increased ($p < 0.05$) by day 3. Experiments were also performed in which measurements were made on animals allowed to recover in air following a 7-day continuous exposure to 1570 μg/m^3 (0.8 ppm) ozone. Prolyl hydroxylase activity returned to control values after 12 to 13 days of recovery. However, hydroxyproline levels remained constantly elevated over 28 days of recovery.

A similar investigation by Hussain et al.[107] was conducted in which rats were exposed continuously to 1570 μg/m^3 (0.8 ppm) ozone for 7 days. Results were generally similar to those reported for 1570 μg/m^3 (0.8 ppm) in the above mentioned study,[108] so that only additional information will be described here. The rates of collagen and noncollagenous protein synthesis increased over the 7-day exposure period. Collagen synthesis was greater than noncollagenous protein synthesis, particularly on days 1 through 4. There was a net accumulation of total lung collagen ($p < 0.05$) by day 3, which remained constantly elevated over the 7 days, reflecting the increased collagen synthesis.

Lung tissue ground substance may include hyaluronic acid, chondroitin sulfuric acids A, B, and C, heparin, keratosulfate, and the proteins collagen and elastin. In one experiment, Buell et al.[23] used four litter-mate rabbits. Two were exposed to 1960 μg/m^3 (1 ppm) ozone for 1 hr, one of which was sacrificed immediately after exposure and the second, 24 hr later. Another was killed immediately after exposure to 9800 μg/m^3 (5 ppm) for 1 hr. The fourth rabbit was used as a control. After enzyme hydrolysis of the protein fraction of the lung tissue, the presence of carbonyl compounds (e.g., aldehydes and ketones) was determined. The carbonyl compounds were identified spectrophotometrically as the corresponding dinitrophenyl-hydrazones. The data from animals sacrificed immediately after exposure were similar to those obtained 24 hr after exposure. It was hypothesized that these carbonyl compounds might cross-link collagen or elastin, leading to structural changes in lung tissue.

Last et al.[118] studied in vitro mucus glycoprotein secretion by tracheal explants from rats exposed to 390 or 1570 μg/m³ (0.2 or 0.8 ppm) ozone for 8 or 24 hr/day for periods of 2 to 90 days. Significant ozone-concentration-related decreases in glycoprotein secretion were observed. The depression of secretion at days 2 to 3 could be reversed by the administration of indomethacin or hydrocortisone during exposure.

Lipids — Many investigators postulate that lipid peroxidation is an important toxic mechanism in ozone injury. These ideas are presented in reviews[39,129] that describe the propensity of ozone to react with the ethylene groups of unsaturated fatty acids. This reaction can lead to free radical formation, which in the presence of molecular oxygen leads to the peroxidation of the unsaturated fatty acid by formation of fatty acid lipoperoxides. Alterations found in erythrocytes following ozone exposure are often cited as evidence for these concepts.[82,86,87,132] (For a more complete discussion of effects on erythrocytes, see Chapter 9.)

Buell et al.[22] suggested that the interaction of ozone and water results in the fomation of atomic- or radical-oxygen and molecular oxygen, whereas Alder and Hill[2] postulated the formation of a variety of radicals. Although high-energy sources such as X-irradiation undoubtedly decompose water into a variety of free radicals, it has not been shown that ozone can form more than one.

That free radicals may be involved in ozone toxicity is suggested indirectly by the work of Mustafa et al.[151] The influence of ozone on superoxide dismutase (SOD) activity of the lung was investigated. SOD is an inducible enzyme that catalyzes the dismutation of superoxide radical, thereby preventing oxidant toxicity that may be consequent to this radical. Rats were exposed to 390, 980, or 1570 μg/m³ (0.2, 0.5, or 0.8 ppm) ozone for 7 days. While it is not stated explicitly, it can be assumed from the abstract that exposure was continuous. SOD activity increased by 17 percent, 26 percent, and 38 percent after exposure to 390, 980, and 1570 μg/m³ (0.2, 0.5, or 0.8 ppm), respectively. At the highest ozone concentration, the location of the SOD activity was determined, and it was found that exposure resulted in a 38-percent increase in the cytosolic fraction and a 46-percent increase in the mitochondrial fraction. Using a different exposure mode, rats were exposed stepwise to 1570 μg/m³ (0.8 ppm) for 72 hr, to 2940 μg/m³ (1.5 ppm) for 24 hr, and finally to 5890 μg/m³ (3 ppm) for 8 hr. Following treatment, SOD activity increased (as ozone concentration increased) by 15 percent, 25 percent, and 50 percent in the cytosol and 50 percent, 67 percent, and 118 percent in the mitochondria, respectively. The authors suggest that the increase in SOD activity represents adaptive changes in the lung that might reduce oxidant toxicity.

Pryor et al.[171] and Pryor[170] exposed pure samples of polyunsaturated fatty acids (methyl linoleate and methyl linolenate) to 0 to 2940 μg/m³ (0 to 1.5 ppm ozone) in vitro and followed the formation of peroxides, conjugated dienes, and thiobarbituric-acid-reactive material as a function of time. Ozone shortened the induction period for auto-oxidation, but did not significantly affect the rate of product formation after the induction period. As the concentration of ozone was raised from 0 to 2940 μg/m³ (0 to 1.5 ppm) during the induction period, there were greatly increased rates of peroxide formation, a slightly increased rate of conjugated diene formation, and no significant change in the rate of production of thiobarbituric-acid-reactive material. When vitamin E was added, the induction period was lengthened, but the rate of product formation during the auto-oxidation phase was unchanged. The authors hypothesized that most of the ozone-induced in vivo alterations occur during the induction period of lipid auto-oxidation.

Roehm et al.[174,176] studied the oxidation of polyunsaturated lipids in vitro by exposing samples of pure methyl esters of fatty acids to 2940 μg/m³ (1.5 ppm) ozone and 2820 μg/m³ (1.5 ppm) NO_2. At equivalent concentrations of these two oxidants, ozone produced the greatest changes. Methyl linoleate was completely reacted by ozone to yield stable ozonides. These researchers hypothesized that ozone acts by direct addition of ozone across the fatty acid double bond, and this agrees with the mechanism proposed by Criegee[41] concerning the primary mechanism for ozonolysis.

Other investigators have studied the effects of ozonides in vitro and in vivo. Menzel et al.[133] injected intradermally 10 picograms to 10 μg of fatty acid ozonides (from oleic, linoleic, linolenic, and arachidonic acids) into animals and found increased vascular permeability of the capillaries, as measured by extravascularly located, pontamine-blue-bound serum proteins. This reaction was blocked by simultaneous antihistamine injection or by prior treatment with compound 48/80 (a substance that can deplete histamine stores). It therefore appeared that histamine was involved in the toxicity of the ozonides (but histamine release could be secondary and not primary). The same series of experiments also showed that the ozonide caused peroxidation of isolated microsomes and mitochondria. There were also indications of oxidation of erythrocyte membranes. Cortesi and Privett[40] also prepared a methyl linoleate ozonide and administered it to rats by intravenous injection or by oral dosage. This treatment was lethal as a result of acute lung edema. Also, significant changes occurred in the fatty acid composition of serum and lung lipids.

To investigate some of the possible effects of ozone on membrane lipids, Teige et al.[196] exposed phosphatidyl choline liposomes containing trapped glucose and human erythrocytes to ozone in vitro (3 μmoles ozone/min). Phosphatidyl choline is a phospholipid commonly found in cell membranes. Ozone exposure caused a leakage of glucose from the liposomes. Liposomes exposed to ozone were more active than ozone alone in causing lysis of red blood cells. Hydrogen peroxide was found not to be responsible for this effect.

Chow and Tappel[32] observed an increased formation of malonaldehyde (a product of lipid peroxidation) in the lungs of rats following continuous exposure to 1370 $\mu g/m^3$ (0.7 ppm) for 5 days or 1570 $\mu g/m^3$ (0.8 ppm) for 7 days. The formation of malonaldehyde, along with the ozone-induced increased activities of glutathione peroxidase and glucose-6-phosphate dehydrogenase (G-6-PD), was partially inhibited as a logarithmic function of dietary α-tocopherol (vitamin E) from 0 to 1500 $\mu g/kg$. Mustafa et al.[148] found no lipid peroxidation (as determined by assay for thiobarbiturate-reacting materials and conjugated dienes) in the lung homogenate of rats exposed to 3920 $\mu g/m^3$ (2 ppm) ozone for 8 hr.

Roehm et al.[174,175] continuously exposed rats to 1960 $\mu g/m^3$ (1.0 ppm) ozone, which eventually resulted in 100-percent mortality due to pulmonary edema. Fifty percent of the α-tocopherol-depleted group died (LT_{50}) after 8.2 days of continuous exposure, while the vitamin-supplemented rats exhibited an LT_{50} of 18.5 days. When Fletcher and Tappel[68] studied the continuous effects on rats of ozone from exposure to 1570 $\mu g/m^3$ (0.8 ppm) or higher concentrations for 7 days, they too found that α-tocopherol protected the animals from severe toxicity (mortality) and weight change. At low levels, the vitamin E protection against lung lipid peroxidation was a reciprocal function of the logarithm of dietary α-tocopherol. Rats maintained on vitamin-E-supplemented diets and exposed to 1560 $\mu g/m^3$ (0.8 ppm) ozone continuously for 7 days were also partially protected from alteration in 6-phosphogluconate dehydrogenase (6-P-GD), 6-phosphogluconate (6-PG), and malic enzyme. Later investigations by Roehm et al.[175] showed that rats continuously exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 9 days showed a significantly shorter LT_{50} and greater symptoms of respiratory distress when they were depleted of vitamin E. After 6 weeks of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone, pulmonary edema and higher mortalities were noted in animals with diets depleted in α-tocopherol. Also, their lung weights were different from those of the α-tocopherol-supplemented groups. After 6 weeks of ozone treatment, significant changes were seen in the fatty acid composition of total lung lipid. The most extensive change was an increase in arachidonic acid in both diet groups ($p < 0.01$), but the increase was higher in the vitamin-E-depleted rats ($p < 0.05$). This effect occurred late in the exposure and it was associated with the onset of pulmonary edema and death. Unlike the lung tissue lipids, the fatty acids from endobronchial saline lavage were little affected by ozone or vitamin E. In both exposures, the onset of the edema was associated with elevated levels of arachidonic acid and docosahexenoic acid (22:6) ($p < 0.05$). These two compounds are important constituents of cell membranes, so membranes could be the site of ozone damage.

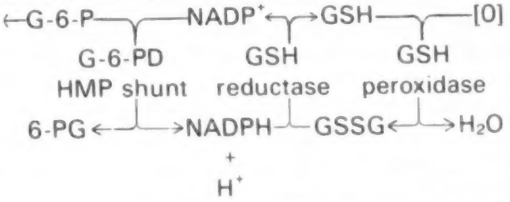
Similar results were obtained by Menzel et al.[132] who exposed rats continuously to ozone. After 3 days of exposure to 2078 $\mu g/m^3$ (1.6 ppm), linoleic and linolenic acid concentrations of lavage lipids were reduced and remained reduced over 17 days of exposure. Lung tissue lipids were also altered in rats exposed for 9 days to 1960 $\mu g/m^3$ (1 ppm) and maintained on diets either supplemented with or deficient in vitamin E. The greatest change was an increase in arachidonic acid that occurred to a greater extent in the animals with vitamin E deficiency. There were also decreases in linolenic acid in both diet groups; however, supplemental vitamin E partially retarded this change. Oleic, stearic, and palmitic acids also were decreased, but only in the vitamin-E-deficient group. Similar measurements were made on rats exposed continuously for 6 weeks to 980 $\mu g/m^3$ (0.5 ppm) ozone. Under these conditions, the greatest changes after ozone exposure were a decrease in oleic acid (particularly in the vitamin-E-deficient group), a decrease in linolenic acid in the vitamin-E-deficient rats, an increase in palmitic acid in the vitamin-E-deficient group, and an increase in the arachidonic acid, particularly in the vitamin-E-deficient animals. Sixty-five days of exposure to 980 μg/m^3 ozone also caused a slight decrease (which appears not to be statistically significant) in lung tissue sulfhydryl content. From day 18 to 65 of a continuous exposure to an unspecified ozone concentration (presumably 980 μg/m^3, or 0.5 ppm) there was a decrease in serum-reduced glutathione in both diet groups.

Menzel et al.[131] also showed that a short exposure of perfused rat lungs to 5880 μg/m^3 (3 ppm) decreased the enzymatic conversion of arachidonic acid to prostaglandins in such a way as to suggest an uncompetitive inhibition of prostaglandin synthetase by ozone. If this response occurs in vivo at lower concentrations of ozone, it would be possible that this could be the mechanism responsible for the ozone-induced increase in arachidonic acid seen in the previously discussed studies [132,175]

Kyei-Aboagye et al.[117] also studied pulmonary lipids. Rabbits exposed to 1960 μg/m^3 (1 ppm) ozone for 4 hr showed a reduced incorporation of ^{3}H-oleate into lecithin ($p = 0.02$). Pulmonary lavage from these animals revealed increased activity of radiolabeled lecithins. The authors observed that ozone may affect the lung by decreasing lecithin formation while simultaneously stimulating the release of surfactant lecithins. The formation of pulmonary phospholipid was investigated by Seto et al.[186] When rabbits were exposed to 980 and 1960 μg/m^3 (0.5 and 1 ppm) ozone for 1 to 2 weeks, the amount of ^{14}C taken into sphingomyelin, lecithin, phosphatidyl inositol plus phosphatidyl serine, and phosphatidyl ethanolamine was significantly reduced. The same researchers studied these phenomena further.[187] Under continuous exposure to 980 μg/m^3 (0.5 ppm) ozone for up to 2 weeks, rabbit lungs showed decreased incorporation of ^{14}C into lecithin until 3 hr post exposure, when this parameter returned to normal. The reduction in phospholipid formation was more pronounced after 2 weeks of exposure rather than 1 week of exposure. An exposure to 1960 μg/m^3 (1 ppm) for 1 hr resulted in a decreased synthesis of sphingomyelin, lecithin, phosphatidyl inositol, phosphatidyl serine, and phosphatidyl ethanolamine.

Even though the lipid composition of pulmonary wash fluid can be altered by ozone exposure, it would appear that the surface-tension-lowering properties of this fluid (which influence pulmonary function) are relatively unaltered. Gardner et al.[81] and Huber et al.[103] reported that exposure of rabbits to 19,600 μg/m^3 (10 ppm) and 9800 μg/m^3 (5 ppm), respectively, did not destroy the surface activity of pulmonary wash fluid. However, Mendenhall and Stokinger[127] exposed saline washings obtained from the lungs of mice exposed to 9800 to 15,700 μg/m^3 (5 to 8 ppm) ozone and noted rapid increases in the film pressure (the force opposing surface tensions). The authors suggested that if analogous changes were to occur in vivo, the consequence would be an increase in the distensibility of the lungs, a situation proposed to be conducive to the development of emphysema. This effect was not confirmed in another study,[8] in which saline washings from the lungs of dogs were exposed in vitro to similar concentrations of ozone.

Sulfhydryl Compounds and Pyridine Nucleotides — A number of intracellular compounds are active in cellular redox reactions and as such can constitute an antioxidant defense system. Many of the effects of ozone on this system have been reviewed. [136,149] Much of the research with ozone has centered on the reduced pyridine nucleotides, namely, reduced nicotinamide adenine dinucleotide (NADH) and reduced nicotinamide adenine dinucleotide phosphate (NADPH), and on sulfhydryl compounds, particularly reduced glutathione (GSH) and related enzymes.[128] The relationship of these compounds to antioxidant activity has been described as follows:



(G-6-P = glucose-6-phosphate; 6-PG = 6-phosphogluconate; G-6-PD = glucose-6-phosphate dehydrogenase; HMP shunt = hexose monophosphate shunt; NADP$^+$ = nicotinamide adenine dinucleotide phosphate; NADPH = reduced NADP; GSH = reduced glutathione; GSSG = oxidized glutathione; [0] = oxidizing moiety [i.e., hydrogen peroxide, free radical, lipid peroxide];

GSH peroxidase = glutathione peroxidase; and GSH reductase = glutathione reductase.)

A number of researchers[128,141,153] have shown that ozonization of aqueous solutions of NADH and NADPH results in their oxidation. Menzel[128] found that after oxidation (0.032 to 0.057 μmole O_3/min x 3 hr), NADH and NADPH were still biologically active as coenzymes. However, Nasr et al.[153] reported that ozone (33 ppm, 1000 ml/min, 50 min) destroyed NADPH but had no effect on $NADP^+$. In vitro exposure[141] to 0.1 to 6 μmoles ozone oxidized NADH, but it had no effect on nicotinamide adenine dinucleotide (NAD). Similar results were not found in vivo[153] when rats were exposed to 64,780 μg/m^3 (33 ppm) ozone for 1 hr. The NADPH/$NADP^+$ ratio of the tracheal epithelium was not significantly altered.

In vitro ozone exposure[128] (0.032 to 0.057 μmole O_3/min x 3 hr) was also able to oxidize aqueous solutions of a number of biologically active reducing substances (cysteine, GSH, and thioglycolic acid). This treatment inactivated papain and glyceraldehyde 3-phosphate dehydrogenase and oxidized GSH and GSSG to non-biologically reducible forms.

The role of sulfur-containing compounds in ozone toxicity is partially illustrated by the work of Fairchild et al.[64] Inhalation of a variety of sulfur compounds (1-hexamethiol, methanethiol, dimethyl disulfide, di-tert-butyl disulfide, benzene thiol, and hydrogen sulfide) partially protected mice from the lethal effects of a 4-hr exposure to 8000 to 12,000 μg/m^3 (4.1 to 6.1 ppm) ozone. The functional unit of the sulfur units appeared to be -SH or SS-, or both, but not -S-, as dimethyl-sulfide and thiophene were not effective.[64]

The content of nonprotein sulfhydryls (NPSH) (a major source of cellular reducing substances), GSH, and GSSG of lung homogenate from ozone-exposed rats was determined by DeLucia et al.[45] The NPSH level was not significantly affected by exposure to 1570 μg/m^3 (0.8 ppm) for up to 24 hr, to 2940 μg/m^3 (1.5 ppm) for up to 8 hr, or to 3920 μg/m^3 (2 ppm) for up to 4 hr. When the animals were exposed to this higher concentration for 6 and 8 hr, however, there was a decrease $(p < 0.01)$ in NPSH. GSH, which represents about 90 percent of the lung NPSH, was significantly reduced at 7850 μg/m^3 (4 ppm) ozone for 6 hr, and GSSG remained unchanged under these conditions. Further experimentation suggested that mixed disulfides were transiently formed that reacted with other sulfhydryl groups of lung tissue. Analysis also indicated that GSH appeared to be the only NPSH bound to pulmonary tissue proteins via a mixed disulfide linkage. In an earlier study, DeLucia et al.[44] found no changes in sulfhydryl levels in lung homogenates of rats continuously exposed (10 days) to 1570 μg/m^3 (0.8 ppm) ozone. However, there was a 20-percent decrease $(p < 0.05)$ in cytochrome C reductase activity and a 32-percent increase $(p < 0.05)$ in G-6-PD activity.

The effects of dietary α-tocopherol on enzyme systems and lipid peroxidation within the lungs of rats exposed continuously to 1370 μg/m^3 (0.7 ppm) for 5 days or 1570 μg/m^3 (0.8 ppm) for 7 days were explored by Chow and Tappel.[32] The increased $(p < 0.05)$ formation of malonaldehyde (a product of lipid peroxidation) and the activities of GSH peroxidase and G-6-PD (see preceding section on lipids) were partially inhibited as a logarithmic function of dietary α-tocopherol (from 0 to 1500 mg/kg). The increased $(p < 0.05)$ activity of GSH reductase was not affected by the level of dietary vitamin E. In addition, the activity of GSH peroxidase and concentration of malonaldehyde were linearly related $(p < 0.001)$. The authors suggest that this represents an apparent compensation mechanism in that, with increased levels of lipid peroxides, there is a corresponding increase in the activity of GSH peroxidase, which in turn increases lipid peroxide catabolism.

The effect of acute (4-hr) and chronic (4-hr/day, up to 30 days) exposure of mice to ozone on lung GSH and vitamin C content was examined by Fukase et al.[74,75] Immediately following the acute exposure to 16,100 μg/m^3 (8.2 ppm) ozone, there was a decrease $(p < 0.05)$ in GSH content. At 7850 μg/m^3 (4 ppm) ozone, there were no changes immediately after exposure. But 2 days later, GSH content increased $(p < 0.1)$ and remained elevated for several days. Acute exposures to various ozone concentrations between 2350 and 16,100 μg/m^3 (1.2 and 8.2 ppm) resulted in a decrease $(p < 0.05)$ in vitamin C content of the lung. Conversely, chronic exposures for 30 days to 390, 980, and 1960 μg/m^3 (0.2, 0.5, and 1.0 ppm) ozone resulted in a progressive increase in GSH and vitamin C content as the ozone concentration and length of exposure (up to 30 days) increased. After 7 days of exposure to 980 or 1960 μg/m^3 (0.5 or 1.0 ppm) ozone, there were increases $(p < 0.05)$ in G-6-PD, GSH reductase, and GSH peroxidase. The administration of vitamin C was found to retard partially $(p < 0.05)$ the ozone-induced (1960 μg/m^3, 4 hr/day, 7 days) increase in G-6-PD.

Chow and Tappel[31] studied the effects of a 7-day continuous in vivo exposure to 1570 $\mu g/m^3$ (0.8 ppm) ozone on hexose monophosphate shunt and glycolytic enzymes of lung homogenates of rats maintained with and without supplemental dietary α-tocopherol. The vitamin E had no effect on the enzyme activities in air-control animals. Ozone caused increases ($p < 0.01$) in G-6-PD and 6-phosphogluconate dehydrogenase (6-P-GD) activities in the lungs of animals from both diet groups, although the increase was less ($p < 0.05$) in those rats that received supplemental vitamin E. Malic enzyme activity was also increased following ozone exposure in the normal ($p < 0.05$) and the supplemented ($p < 0.1$) diet groups, but the increase was less ($p < 0.05$) in the supplemented vitamin E group. Neither ozone nor α-tocopherol supplementation altered the activity of soluble malic dehydrogenase. The enzymes that regulate glycolysis—phosphofructokinase and pyruvate kinase—had increased activities following ozone exposure and were not affected by vitamin E in the levels administered. Ozone exposure resulted in increases in aldolase activity ($p < 0.1$) in the dietary-supplemented animals, but not in the normal diet group ($p < 0.1$) compared to air controls. The differences between the supplemented and normal diet groups receiving ozone were not significant. Lactate dehydrogenase (LDH) activity increased in both diet groups following ozone exposure ($p < 0.001$), although the increase was less ($p < 0.1$) in the supplemental diet group.

To determine the influence of the duration of ozone exposure on some of these ozone-induced responses, rats maintained on normal diets were exposed continuously to 1470 $\mu g/m^3$ (0.75 ppm) ozone.[31] The activities of GSH peroxidase, GSH reductase, G-6-PD, 6-P-GD, and pyruvate kinase were decreased after 1 day of exposure. Thereafter, they increased until day 10. By day 30, all enzyme activities except for GSH peroxidase and GSH reductase had decreased from their 10-day values but were still increased over control values. GSH reductase exhibited a very slight decrease between days 10 and 30. GSH peroxidase continued to increase over this time period. The authors hypothesized that an increased rate of glycolysis and hexose monophosphate shunt activity is an adaptive response to the increased need for reductive detoxification of ozone-induced lipid peroxidation.

Dungworth et al.[50] exposed monkeys to ozone for 8 hr/day for 7 days. Six rhesus monkeys received 1570 $\mu g/m^3$ (0.8 ppm), and six were exposed to 980 $\mu g/m^3$ (0.5 ppm). Bonnet monkeys (2 to 4 per exposure group) were exposed to either 980 $\mu g/m^3$ (0.5 ppm), 690 $\mu g/m^3$ (0.35 ppm), or 390 $\mu g/m^3$ (0.2 ppm). In the lungs of ozone-exposed monkeys, there were increased activities of GSH peroxidase, GSH reductase, G-6-PD, NADPH-cytochrome-C reductase, succinate oxidase, acid phosphatase, and β-N-acetyl-glucosaminidase. Linear regression analysis indicated that there was a significant ($p < 0.05$) correlation between ozone concentration and increase in enzyme activity in both rhesus and Bonnet monkeys. Morphological investigations of the same animals (see later section on morphology studies) showed the presence of a lesion at the level of the respiratory bronchiole and increased numbers of Type 2 cells.

Chow et al.[28] exposed rats to 390, 980, or 1570 $\mu g/m^3$ (0.2, 0.5, or 0.8 ppm) ozone continuously (8 days) or intermittently (8 hr/day × 7 days) before biochemical assays of lung homogenates. For the continuous exposure to the two higher ozone concentrations, activities of GSH peroxidase, GSH reductase, and G-6-PD were increased ($p < 0.05$) over controls. At the lower ozone concentration, there was an increase ($p < 0.05$) in the activities of GSH peroxidase and GSH reductase. Linear regression analysis indicated that there was a linear increase in all 3 enzyme activities as the concentration of ozone was increased ($p < 0.001$). Similar results were obtained for the intermittent exposure groups. No statistical comparisons of the intermittent and continuous exposure groups were made. The authors suggest that the increase in enzyme activity is in response to the need for reduction of ozone-induced lipid peroxides.

Similar measurements were made by Mustafa and Lee[150] of the lungs of rats and monkeys exposed to various concentrations of ozone. Lung homogenates of rats exposed continuously for 7 days to 1570 $\mu g/m^3$ (0.8 ppm) were examined. On the first day, the activities of microsomal NADPH-cytochrome-C reductase and cytosolic G-6-PD activities were slightly depressed, but by day 2 they were elevated, reaching a plateau at days 3 to 4. By day 7, NADPH-cytochrome-C reductase had increased by 42 percent ($p < 0.001$), and G-6-PD by 67 percent ($p < 0.001$). Similar increases were found in O_2 consumption and mitochondrial-succinate-cytochrome-C reductase (see the following section on mitochondrial enzyme activities).

The influences of intermittent (8 hr/day) and continuous exposure for 7 days were compared as part of the same investigation by Mustafa and Lee.[180] Exposures were to 390, 980, or 1570 μg/m³ (0.2, 0.5, or 0.8 ppm) ozone. Specific data were not given for NADPH-cytochrome-C reductase or G-6-PD; however, the authors stated that these enzyme activities increased in a manner similar to succinate oxidase activity (see the following section on mitochondrial enzyme activities). Thus it would seem that rats exhibited the greatest significant increase, with effects being observed at 390 μg/m³ (0.2 ppm), while these enzyme activities in rhesus monkeys only increased significantly ($p < 0.05$) after exposure to 1570 μg/m³ (0.8 ppm). Bonnet monkeys exhibited a significant ($p < 0.05$) increase in these enzyme activities at 690 μg/m³ (0.35 ppm).

Biochemical assays of the lungs of rats and rhesus monkeys were also made by Chow et al.[30] Assays were made following a 7-day exposure (8 hr/day) to 980 μg/m³ (0.5 ppm) ozone. In rat lung homogenates, there were 20- to 26-percent increases ($p < 0.05$) in the activities of GSH peroxidase, GSH reductase, and G-6-PD. GSH was also significantly elevated by 22 percent. Though increases in all these parameters occurred in monkeys (n=12), the increases (which ranged from 10 to 15 percent) were smaller in magnitude as compared to the rats, and they were not statistically significant because of relatively large variations. Similar measurements were made on the erythrocytes of these animals, but no significant changes were observed.

The effects of intermittent (8 hr/day) and continuous ozone exposures for 7 days on rats were compared by Schwartz et al.[184] Ozone exposures were to 392, 980, or 1568 μg/m³ (0.2, 0.5, or 0.8 ppm). Biochemical changes were compared to morphologic alterations. The latter are discussed in more detail in a later section on morphology studies. The activities of G-6-PD and NADPH-cytochrome-C reductase in cellular oxygen utilization (succinate oxidase activity) were increased in an ozone-concentration-dependent fashion following exposure. The increases in the continuous exposure groups were only slightly greater than in the intermittently exposed animals. Concurrent morphologic studies also showed little difference between continuous and intermittent exposure. Therefore it would appear the recovery did not occur during the clean air period of the intermittent exposure.

Chow et al.[29] examined the biochemical responses of lung homogenates of ozone-exposed rats during recovery and re-exposure. All ozone exposures were continuous (3 days) to 1568 μg/m³ (0.8 ppm). The activities of GSH peroxidase, GSH reductase, and G-6-PD were significantly ($p < 0.01$) increased by 25, 18, and 46 percent, respectively, immediately after the initial 3-day exposure. There was also an 85-percent increase ($p < 0.001$) in protein synthesis and a 24-percent increase ($p < 0.001$) in nonprotein sulfhydryl (NPSH) content at this time. These biochemical changes began to revert toward control values after a 2-day holding period in clean air. Recovery continued and completely reached control values 9 days after exposure cessation. No further changes were found up to 30 days after ozone exposure ended. The ozone-exposed rats were re-exposed to ozone (1568 μg/m³, or 0.8 ppm, continuously for 3 days) at various times during the recovery period (day 6, when recovery was incomplete, and days 13 and 27). Following this re-exposure, values from all biochemical measurements increased to approximately the same levels as found with the initial ozone exposure, thereby indicating that no adaptation to ozone occurred.

The effects of ozone tolerance on many of these enzyme systems were investigated by Chow.[27] To develop tolerance, rats were exposed to 0 or 1490 μg/m³ (0.76 ppm) ozone for 3 days. The ozone treatment resulted in increased activities of GSH peroxidase, GSH reductase, and G-6-PD, and in GSH levels that returned to normal approximately 7 days after recovery in air. After 8 days of recovery in clean air, some animals were exposed to 7840 μg/m³ (4 ppm) ozone for 8 hr. Lung homogenates were assayed 16 hr later. Fifty percent of the non-pre-exposed (i.e., nontolerant) rats died following the 7840 μg/m³ (4 ppm) exposure, but only 1 of 7 of the tolerant group died. The lungs of the tolerant and nontolerant animals exposed to the high ozone dose exhibited an increase in protein content, decreased ($p < 0.05$) activities of GSH peroxidase, GSH reductase, and G-6-PD, and a decrease in GSH levels. However, the lungs of tolerant rats had significantly greater ($p < 0.05$) enzyme activities and higher GSH levels compared to nontolerant animals. When the animals were allowed to recover for 9 days before the 7840 μg/m³ (4 ppm) challenge, there were no differences between the tolerant and nontolerant groups. In a similar study, rats that were 10 days younger were pre-exposed for 3 days to 1568 μg/m³ (0.8 ppm) before an 8-hr challenge with 7644 $\mu g/m^3$ (3.9 ppm) ozone. Allowing an 11-day recovery period between the two exposures resulted in findings similar to those for the 8-day recovery. The authors suggest that the tolerance observed might render the animals more resistant to peroxidative, ozone-induced damage.

Mitochondrial Enzyme Activities — Mustafa[147] demonstrated alterations in lung mitochondrial oxygen consumption in lung homogenates of rats exposed to 5880 $\mu g/m^3$ (3 ppm) ozone for 4 hr and then placed in air for several days. Oxidation of succinate was decreased ($p < 0.05$) immediately after exposure and remained so during the next 12 hr. The rate of oxidation reached control values by 24 hr and then increased and peaked ($p < 0.001$), relative to control, between 72 and 96 hr. Oxygen consumption then began to return to normal values and was not significantly different from control by day 21. These observations made with succinate are similar to those made using 2-oxyglutarate, glycerol-1-phosphate, and ascorbate-Wurster's blue as substrates, except that the magnitude of the changes was different. Succinate-cytochrome-C reductase activity was greater in the exposed lungs after recovery for 2 to 4 days. When the mitochondrial fraction of the lungs was examined, generally similar changes in O_2 consumption were observed. The increase in oxidation was found up to day 7 of recovery, but not after days 14 and 21 of recovery. To elucidate the reasons for the observed alterations in oxidative activity, other experiments were performed. The cytochrome content of mitochondria (on a per-milligram-of-protein basis) was not significantly different in the exposed animals when examined after 2, 3, and 4 days of recovery. However, the mitochondrial population of the exposed animals increased and was significantly higher ($p < 0.01$) after 48 hr of recovery. After 7 days, the population began to decline. Concurrent transmission electron microscopic observations indicated that there appeared to be no increase in number of mitochondria within the cells of the exposed animals' lungs, but the number of Type 2 cells, which contain numerous mitochondria, had increased by 50 to 100 percent after ozone exposure, and succinate-O_2 consumption of these cells was unchanged (on a per-milligram-of-protein basis).

In contrast to the inhibitory effects of acute exposures to high concentrations of ozone, low-level, chronic exposures result in an increase of lung oxidative metabolism. Mustafa et al.[144] studied mitochondrial oxygen consumption in rats exposed continuously to 1568 $\mu g/m^3$ (0.8 ppm) for 10 to 20 days. They found a 45-percent increase ($p < 0.02$) in oxygen utilization by the 20th day of the experiment. There was also a 20- to 30-percent increase in the oxidation of 2-oxyglutarate and glycerol-1-phosphate. After 10 days of exposure, there was a simultaneous increase (threefold) ($p < 0.01$) in the number of Type 2 alveolar cells that are rich in mitochondria, so perhaps the increase in O_2 comsumption was due to proliferation of these cells. After 7 days of exposure to 392, 980, or 1568 $\mu g/m^3$ (0.2, 0.5, or 0.8 ppm) ozone, there were increases ($p < 0.05$) in O_2 consumption of 17, 30, and 40 percent, respectively.

The effects of various modes of ozone exposure on O_2 consumption of lung tissue were investigated by Mustafa and Lee.[150] In the first experiment, rats were exposed to 1568 $\mu g/m^3$ (0.8 ppm) continuously for 1 to 30 days. The rates of oxygen consumption for oxidation of 2-oxyglutarate, succinate, and glycerol-1-phosphate were slightly reduced in lung homogenate at day 1, but began to increase thereafter. The increased ($p < 0.001$) rate of O_2 consumption reached a peak at day 4 and plateaued over the remainder of the exposure (up to 30 days). The oxidation of succinate was increased more than the other two substrates. During a 7-day continuous exposure to this ozone concentration (1568 $\mu g/m^3$ or 0.8 ppm), there was an initial decrease (day 1) and a subsequent increase (day 2) in the activity of mitochondrial-succinate-cytochrome-C reductase that plateaued between days 3 and 7. By day 7, the increased activity was 55 percent above control ($p < 0.001$). Similar results were found for microsomal NADPH-cytochrome-C reductase and G-6-PD (see preceding section on sulfhydryl compounds and pyridine nucleotides).

As part of the same study,[150] the effects of continuous and intermittent (8 hr/day) exposure of rats for 7 days were compared at the end of the exposure. Exposures were to 392, 980, or 1568 $\mu g/m^3$ (0.2, 0.5, or 0.8 ppm) ozone. The increases in O_2 consumption (using succinate as a substrate) and succinate-cytochrome-C reductase activity were all significant ($p < 0.05$) and fairly proportional to the ozone concentration. At the lower ozone concentration, the increase in enzyme activity was slightly greater in the intermittent exposure group. However, the reverse occurred at the two higher concentrations. The differences in the data from the two modes of exposure (i.e., intermittent versus continuous) were not significant. Similar concentration-related responses occurred for NADPH-cytochrome-C reductase and G-6-PD (see preceding section on sulfhydryl compounds and pyridine nucleotides). Monkeys were exposed intermittently to various concentrations of ozone, and after 7 days, enzyme activities of lung tissue were made. Rhesus monkeys (n=24) showed elevations in succinate oxidase activity of 12 percent (not statistically significant) at 980 $\mu g/m^3$ (0.5 ppm) and of 24 percent ($p < 0.05$) at 1568 $\mu g/m^3$ (0.8 ppm). Greater increases were observed in Bonnet monkeys (n=13): 13 percent (not statistically significant), 22 percent ($p < 0.05$), and 30 percent ($p < 0.02$), for 392, 686, and 1568 $\mu g/m^3$ (0.02, 0.35, and 0.8 ppm), respectively. However, greater increases were observed in the rats for the corresponding ozone concentrations. Similar species differences with respect to ozone concentration were found for NADPH-cytochrome-C reductase and G-6-PD. After 7 days of exposure of the rats, there were significant ($p < 0.05$) increases in NADPH-cytochrome-C reductase and G-6-PD that were related to ozone concentration. Though there was less increase in the intermittent group, there was no statistically significant difference between intermittent and continuous exposure. Similar results were observed for succinate oxidase and succinate-cytochrome-C reductase (see preceding section on sulfhydryl compounds and pyridine nucleotides).

As part of the same study, Mustafa and Lee[150] also examined the effect of vitamin E on the effect of ozone on succinate-dependent O_2 consumption of lung homogenate. Five weeks before ozone exposure, rats were fed diets with either 66 ppm or 11 ppm of vitamin E. The authors state that 66 ppm vitamin E is approximately twice the recommended daily allowance, and that 11 ppm vitamin E is the average concentration in American diets. The animals were then exposed continuously for 7 days to either 196 or 392 $\mu g/m^3$ (0.1 or 0.2 ppm) ozone before assays.The vitamin E-supplemented group (66 ppm) exhibited an increase in succinate oxidase activity of 3 percent (not significant) and 18 percent ($p < 0.05$) at 196 and 392 $\mu g/m^3$ (0.1 and 0.2 ppm) ozone, respectively. Those animals that received 11 ppm vitamin E had greater increases in succinate oxidase activity, namely a 25-percent ($p < 0.02$) and a 38-percent ($p < 0.01$) increase for 196 and 392 $\mu g/m^3$ (0.1 and 0.2 ppm) ozone, respectively. Vitamin E did not cause any significant differences in succinate oxidase activity of control animals. Mustafa and Lee[150] primarily attribute the increases in these enzyme activities to a concomitant increase of Type 2 cells (see the later section on morphology studies), which are rich in mitochondria and exhibit high metabolic activity.

The effects of recovery from and re-exposure to ozone on O_2 consumption of lung was investigated by Chow et al.[29] Rats exposed continuously to 1568 $\mu g/m^3$ (0.8 ppm) ozone for 3 days exhibited a 48-percent increase ($p < 0.001$) in the rate of mitochondrial succinate oxidation. After the animals recovered for 2 days in filtered air, this value began to return to normal and reached control values 9 days after the initial exposure ceased. When the rats were re-exposed to the same ozone concentration (3 days, continuous) on days 6, 13, and 27 of recovery, the rate of mitochondrial succinate oxidation again increased to levels similar to those observed for the intial ozone exposure. Similar effects were observed for GSH peroxidase, GSH reductase, and G-6-PD (see preceding section on sulfhydryl compounds and pyridine nucleotides). So again the data support the inference that adaptation to these ozone-induced responses did not occur.

Other Biochemical Alterations — In a review, Mustafa et al.[149] present the results of a study on the effects of ozone on glucose metabolism in the lung. Rats exposed to 1568 $\mu g/m^3$ (0.8 ppm) ozone continuously for 4 days exhibited a 59 percent ($p < 0.02$) increase in the rate of glucose consumption. In addition, the rates of pyruvate and lactate production both increased 43 percent ($p < 0.02$), but the absolute amount of lactate produced (22.2 μmoles/hr per lung) was greater than the amount of pyruvate produced (3.7 μmoles/hr per lung).

This review[149] also describes an effect of ozone on monoamine oxidase (MAO), an enzyme that catalyzes the metabolic degradation of biological amines. Acute exposure (8 hr) to 3920 $\mu g/m^3$ (2 ppm) ozone caused a 30- to 40-percent ($p < 0.05$) decrease in MAO activity of lung homogenate, mitochondria, and microsomes. However, a 7-day continuous exposure to 1568 $\mu g/m^3$ (0.8 ppm) resulted in a 20- to 35-percent ($p < 0.05$) increase of MAO activity.

Other microsomal enzymes, namely mixed function oxidases like cytochrome P_{450} (cyt. P_{450})

can be affected by ozone. Goldstein et al.[88] found a decrease ($p < 0.01$) in lung microsomal cyt. P_{450} levels immediately or 1, 2, 3, or 5 days after rabbits were exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 90 min. The data for up to 45 days post exposure were fitted to a parabolic curve with a significant ($p < 0.001$) multiple correlation coefficient. The greatest depression of cyt. P_{450} occurred at 3.6 days after ozone exposure. Liver cyt. P_{450} levels from the ozone-exposed rabbits were not significantly different from control immediately or 1 day post exposure. In vitro exposure (1960 $\mu g/m^3$, or 1 ppm, for 90 min) of lung homogenate resulted in a 52-percent decrease in microsomal cyt. P_{450} levels. Lung microsomal fractions similarly treated exhibited a 68-percent decrease in cyt. P_{450}.

A cyt.-P_{450}-dependent mixed-function oxidase (benzpyrene hydroxylase) was also examined for ozone susceptibility. Palmer et al. made measurements of both the lung parenchyma[161] and tracheobronchial mucosa[160] of Syrian golden hamsters exposed to 1470 $\mu g/m^3$ (0.75 ppm) ozone for 3 hr. The lung tissue itself had a 33-percent reduction in the activity of benzpyrene hydroxylase, whereas the tracheobronchial mucosa exhibited a 53-percent drop immediately after exposure. The mucosal enzymatic activity had recovered by 24 hr after ozone exposure.

Lysosomes (cellular organelles containing a variety of enzymes) are also affected by ozone exposure, as several researchers have shown. For example, rats exposed to 1568 $\mu g/m^3$ (0.8 ppm) ozone continuously for 8 days showed significant increases in lysozyme activity in lung homogenate and plasma over the level of lysozyme activity in rats exposed in the same manner to 0, 392, or 980 $\mu g/m^3$ (0, 0.2, or 0.5 ppm) ozone. When animals were exposed to the same ozone concentrations intermittently (8 hr/day for 7 days), there were no significant changes in pulmonary lysozyme activity.[28] In another study,[46] rats were continuously exposed to 1372 $\mu g/m^3$ (0.7 ppm) ozone for 5 days or 1568 $\mu g/m^3$ (0.8 ppm) for 7 days, and the activities of various lysosomal hydrolases were measured in whole lung homogenates and found to increase significantly ($p < 0.05$). The authors attributed this response to an infiltration of phagocytic cells (which have a high concentration of lysosomes) into the lung in response to ozone. The higher enzymatic levels also possibly reflect greater cell membrane lability. The authors hypothesized that the increase in protease and peptidase activities could lead to chronic obstructive pulmonary disease.[46] Although these data are contrary to that of Hurst et al.[105,106] (see the alveolar macrophage section), the authors state that the difference was probably because Hurst used isolated cells (alveolar macrophages), whereas they used whole lung tissue. Damage to lysosomal membranes was also proposed by others after histo- and cyto-chemical studies of the effects of 1372 to 1568 $\mu g/m^3$ (0.7 to 0.8 ppm) ozone administered continuously for 7 days.[25] These researchers observed increased acid phosphatase activity and no changes in the β-glucuronidase activity in alveolar macrophages of rats. The terminal airway epithelium and adjacent structures also exhibited increased acid phosphatase activity. This particular enzyme had a nonvacuolar, as well as an intracellular distribution, indicating lysosomal membrane damage. In another publication, the same authors[26] examined similarly exposed rats for enzyme activities. In areas of the bronchiolar epithelium that were infiltrated by mononuclear cells, there was a lower NADH- and NADPH-diaphorase activity and an increased ATPase activity. However, intra-alveolar septa in the centriacinar region had increased diaphorase, LDH, ATPase and cytochrome oxidase activities. These increases were also seen in septa that were not thickened or infiltrated with increased numbers of cells. It was proposed that these enzymatic changes are part of protective-adaptive mechanisms that operate mainly in the centriacinar region of the lung.

MORPHOLOGY STUDIES

Scheel et al.[181] provided histopathologic evidence of injury caused by a single acute exposure to 1960 or 6272 $\mu g/m^3$ (1.0 or 3.2 ppm) ozone for 4 hr in mice and by repeated intermittent exposures of 15,680 to 88,200 $\mu g/m^3$ (8 to 45 ppm) ozone for 1 hr in rabbits. Pulmonary edema was not observed in mice sacrificed immediately after exposure to 1960 $\mu g/m^3$ (1 ppm) ozone, but moderately engorged capillaries containing an excess of leukocytes were visible. Mice examined 20 hr after exposure showed mild edema and migration of leukocytes into the alveolar spaces. Superficial desquamation of the epithelium in the bronchi and bronchioles was also observed. Inhalation of 6272 $\mu g/m^3$ (3.2 ppm) ozone produced grossly visible edema. The perivascular lymphatic vessels were distended and filled with edema fluid and precipitate. Hyperemia, mobilization of leukocytes, and varying degrees of extravasation of red cells into the tissues accompanied the edema. Sheets of desquamated bronchiolar epithelial cells were seen in the lumina.

Pulmonary lesions observed in cats exposed to 510, 980, and 1960 $\mu g/m^3$ (0.26, 0.5, and 1 ppm) ozone for 4 to 6 hr indicated that desquamation of the ciliated epithelium was apparently dose-related.[15,16] Other ultrastructural changes were cytoplasmic vacuolizations of the ciliated cells that occurred in airways predominantly 0.8 to 1.7 mm in diameter. At the same sites, condensed mitochondria with abnormal cristae appeared. In the same reports,[15,16] swelling and desquamation of Type 1 alveolar cells, swelling and breakage of capillary endothelium, and erythrocyte lysis within the interalveolar capillaries were seen.

Mice (18- to 20-month-old males) were exposed for 6 hr to 980 $\mu g/m^3$ (0.5 ppm), 2160 $\mu g/m^3$ (1.2 ppm), and higher levels of ozone and then were given injections of tritiated thymidine that labeled alveolar cells undergoing DNA synthesis.[59] Inhibition of DNA synthesis occurred at all ozone levels to a similar extent, thus representing maximum inhibition. This depression was seen up to 24 hr after the ozone-exposed mice were transferred to clean air and did not return completely to normal until 72 hr later.[59]

Evans et al.[57,58,60] studied the kinetics of alveolar cell division in rats exposed continuously for 8 days to 686 or 980 $\mu g/m^3$ (0.35 or 0.5 ppm) ozone. Cell proliferation increased to a maximum at 2 days and began to decrease toward normal by the fourth day. The principal dividing cell observed was the Type 2 cell. This process is known to be a mechanism for replacing damaged Type 1 cells. After the fourth day, no further tissue damage or increase in proliferation occurred, suggesting that the tissue had become tolerant to that particular concentration of ozone. To evaluate the extent of this possible tolerance, after 4 days of exposure to 686 $\mu g/m^3$ (0.35 ppm), the ozone concentration was elevated to 980 or 1372 $\mu g/m^3$ (0.5 or 0.7 ppm) for up to 4 days. Under these conditions, morphological damage increased, indicating that tolerance did not occur. However, in rats exposed initially for 4 days to 980 $\mu g/m^3$ (0.5 ppm) and re-exposed for 4 days to 1470 or 1960 $\mu g/m^3$ (0.75 or 1 ppm), some tolerance was evident. Note that studies with young and old mice exposed to ozone have shown a decrease in cell proliferation.[58,190] This result may be due to age, species, or the ozone concentration used in the studies.

From the studies of Stephens et al.[190] conducted with rats, it also appears that the Type 1 cells are the alveolar epithelial cells most sensitive to low levels of ozone, 980 $\mu g/m^3$ (0.5 ppm) for 2 hr. The earliest change noted was a swelling of the mitochondria of these cells, particularly those located in the first two or three alveoli immediately beyond the terminal bronchiolar epithelium. This was followed by more severe alterations, which led to leaving the basement lamina devoid of an epithelial covering. Type 2 cells were resistant to the ozone treatment, and after 4 to 6 hr of exposure, showed signs of spreading over the injured area.

Evans et al.[58] examined renewal of the terminal bronchiolar epithelium of rats at various time periods after a 24-hr exposure to 1372 $\mu g/m^3$ (0.7 ppm). One hour after conclusion of the exposure, almost all proliferating cells were nonciliated; however, by 4 days, only approximately 76 percent of the proliferating cells were nonciliated, the remainder being ciliated. At 15 days, approximately the same relationship was observed. These events as well as other observations led the authors to the conclusion that nonciliated cells divide after ozone exposure and form new ciliated and nonciliated cells. In this manner, the nonciliated cells were thought to act as progenitor cells that participate in the recovery of the damaged terminal bronchiolar epithelium.

Morphologic observations made by Dungworth and coworkers,[49,50] indicate that the rat and Bonnet monkey (*Macaca radiata*) are approximately equal in susceptibility to short-term effects of ozone. Mild but significant lesions were caused in both species by exposure to 392 $\mu g/m^3$ (0.2 ppm) for 8 hr/day for 7 days. The authors stated that detectable morphological effects in the rat occurred at levels as low as 196 $\mu g/m^3$ (0.1 ppm). In both species, the lesion occurred at the junction of the small airways and the gaseous exchange region. In rats, the prominent features were accumulation of macrophages, replacement of necrotic Type 1 epithelial cells with Type 2 cells, and damage to ciliated and nonciliated Clara cells. The principal site of damage was the alveolar duct. In monkeys, the prominent ozone-induced injury was limited to the small airways. At 392 $\mu g/m^3$ (0.2 ppm), the lesion was observed at the proximal portion of the respiratory bronchioles. As concentrations of ozone were increased up to

1568 $\mu g/m^3$ (0.8 ppm), the severity of the lesion increased, and the damage extended distally to involve the proximal portions of the alveolar duct.

Mellick and coworkers[126] found similar but more pronounced effects when rhesus monkeys (3 to 5 years of age) were exposed to 980 to 1568 $\mu g/m^3$ (0.5 and 0.8 ppm) ozone, 8 hr/day for 7 days. In these experiments, the respiratory bronchioles were the most severely damaged, and more distal parenchymal regions were unaffected. Major effects were hyperplasia and hypertrophy of the nonciliated bronchiolar epithelial cells and the accumulation of macrophages intraluminally. In mice, continuous exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone caused nodular hyperplasia of Clara cells after 7 days of exposure.

Similar findings were reported by Schwartz[183] and Schwartz et al.,[184] who exposed rats to 392, 980, or 1568 $\mu g/m^3$ (0.2, 0.5 or 0.8 ppm) ozone for 8 or 24 hr/day for 1 week. Changes observed within the proximal alveoli included infiltration of inflammatory cells and swelling and necrosis of Type 1 cells. In the terminal bronchiole, the changes reported were shortened cilia, clustering of basal bodies in ciliated cells suggesting ciliogenesis, and reduction in height or loss of cytoplasmic luminal projection of the Clara cells. Effects were seen at ozone concentrations as low as 392 $\mu g/m^3$ (0.2 ppm). A dose-dependent pulmonary response to the three levels of ozone was evident. No differences were observed in morphologic characteristics of the lesions between rats exposed continuously and those exposed intermittently for 8 hr/day.

Using electron microscopy, Bils[11] studied the lungs of mice of different ages (4 days or 1 to 2 months) exposed to 1176 to 2548 $\mu g/m^3$ (0.6 to 1.3 ppm) ozone for 6 to 7 hr/day for 1 to 2 days and noted swelling of the alveolar epithelial lining cells without intra-alveolar edema. Swelling of endothelial cells and occasional breaks in the basement membrane were seen. The younger mice exposed for 2 days were the most sensitive, although these observations are contrary to the demonstrations of the limited sensitivity of newborn rats exposed from birth to NO_2.[71,121]

Brummer et al.[21] used scanning electron microscopy (SEM) to examine centriacinar regions of the lung and to count the influx of cells within the lumina in order to evaluate the cellular response during ozone exposure. Continuous exposure to 981 or 1570 $\mu g/m^3$ (0.5 or 0.8 ppm) for 7 days resulted in an 18-fold increase in inflammatory cells within the lumina of proximal alveoli.

Sato et al.[180] studied the effects of 588 $\mu g/m^3$ (0.3 ppm) ozone (3 hr/day for 16 days) on the morphologic features of the conducting airways and alveoli of rats using SEM. Observation of the luminal surface of the bronchi, bronchioles, and terminal bronchioles of the exposed animals showed cilia that were swollen and adhered to one another. Small, smooth-surfaced round bodies were observed mainly around the tips of the cilia. The ciliated areas of the lobar bronchi and proximal bronchioles were covered with a pseudomembrane. Clara cells were more prominent and greater in size in all rats exposed to ozone. The surface of the alveolar ducts and alveolar walls showed scattered areas of cytoplasmic swelling and attachment of round bodies throughout the parenchyma. Alveolar pores were distorted and smaller in size than the control groups. The Type 1 cells, as others have shown, appeared to be most vulnerable to ozone. These studies and others[126] also showed that the vitamin-E-deficient rats were more sensitive to ozone than rats fed a diet supplemented with vitamin E.

Stokinger et al.[195] reported that chronic bronchitis, bronchiolitis, and emphysematous and fibrotic changes develop in the lung tissues of mice, rats, hamsters, and guinea pigs exposed 6 hr/day, 5 days/week for 14.5 months to a concentration slightly above 1960 $\mu g/m^3$ (1 ppm) ozone.

Rats exposed for 3 to 5 months to 1568 $\mu g/m^3$ (0.8 ppm) ozone develop a disease that resembles emphysema, and they finally die of respiratory failure.[192] Ozone results in a greater response of fibroblasts in the lesion, thickening of the alveolar septae, and an increase in number of alveolar macrophages in the proximal alveoli.

Dogs were exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 8 hr daily for 18 months.[72] The resulting damage appeared roughly proportional to the time and concentration of the ozone exposure. The number of alveolar macrophages increased, fibrous elements were deposited, and the small airway lumina were reduced by a thickening of the terminal airways and respiratory bronchiolar walls. The peribronchiolar area contained excessive numbers of lymphocytes and plasma cells, and squamous metaplasia of the columnar and cuboidal epithelium developed.

Another long-term experiment was undertaken by Freeman et al.[70] in which rats were continuously breathing ozone at either 1058 or 1725 μg/m^3 (0.54 or 0.88 ppm) for periods of up to 6 months. When the animals were exposed at the lower ozone level for 2 hr, no immediate significant histological changes were seen; but after 24 hr, epithelial hypertrophy was found between the distal portions of the terminal bronchioles and the alveolar ducts. The peribronchiolar alveolar area contained amorphous material resembling necrotic cells. After 48 hr of exposure, the hypertrophy of the epithelium of the alveolar ducts was in sharp contrast to the controls, and numerous mitotic figures were seen in the terminal bronchiolar and proximal alveolar epithelium. The interstitial tissue in this area contained some cells with pyknotic nuclei. Macrophages were prominent. Fibroblasts appeared to be present beneath the ductal epithelium, and the respiratory bronchiolar epithelium had deposits of connective tissue elements. Histochemical examination suggested a change in the collagen. Additional rats were studied at 6 days, 8 days, 3 weeks, and 6 months, and progressive changes occurred in the airway epithelium. Metaplasia of cuboidal epithelium was seen, which continued to be replaced by connective tissue. However, the terminal bronchioles appeared more normal after 3 weeks of ozone and remained so for the rest of the exposure period. Even with this partial recovery, the deposition of connective tissue interrupted the continuity of the small airways with their proximal alveoli.

When the rats were exposed to 1725 μg/m^3 (0.88 ppm) ozone, epithelial injury was seen in 4 hr. The morphological alterations found at the lower concentration were present in these rats also, but they were more extensive and occurred earlier. Furthermore, after 48 hr of ozone, the bronchiolar epithelium was often metaplastic. By day 3, there was an onset of early fibrosis, which constricted some terminal airways. After 6 days, adenoma-like structures containing large numbers of macrophages were more prevalent. Fibrosis had extended into the terminal bronchioles. After 3 months of exposure to 1725 μg/m^3 (0.88 ppm) ozone, emphysema-like lesions had developed. This study also included a group of animals exposed to both 1692 μg/m^3 (0.9 ppm) NO_2 and 1764 μg/m^3 (0.9 ppm) ozone, and another group that received 4900 μg/m^3 (2.5 ppm) NO_2 and 490 μg/m^3 (0.25 ppm) ozone. They, too, were examined for pathological changes. However, the authors found no synergism because ozone appeared to be largely responsible for the alterations seen at these concentrations.[70]

Investigations by others have supported previously discussed data and, in some cases, have added more information. P'an et al.[162] exposed rabbits to 784 μg/m^3 (0.4 ppm) ozone for 6 hr a day, 5 days a week for 10 months. The authors observed both emphysematous and vascular lesions in the lungs. The small pulmonary arteries were thicker than in controls because of the increased size of the tunica media vasorum, which in some instances appeared to be a result of tissue edema or muscular hyperplasia.

The possibility that daily ozone exposure might increase the incidence of tumors has also been studied. Stokinger[193] reported that acceleration of lung tumorigenesis (adenoma) in a strain of mice susceptible to such tumors occurred from daily ozone exposures to about 1960 μg/m^3 (1 ppm). At 15 months, a tumor incidence of 85 percent was found in the ozone-exposed, as against 38 percent in the control mice. The average number of tumors per exposed mouse was 1.9 compared with 1.5 in the controls. Experimental details were not given.

Kotin and coworkers[114,116] studied the experimental induction of pulmonary adenomas in Strain A mice (which are prone to develop pulmonary adenomas) and in C-57 black mice exposed for 52 weeks to an atmosphere of ozonized gasoline. The concentration of oxidants varied from 1 to 3.8 ppm. The concentration of other pollutants present was not reported. A significantly greater incidence of tumors occurred in the exposed than in the control mice. There was no significant difference in tumor incidence between males and females. The respiratory epithelium of the mice revealed significant hyperplastic and metaplastic responses.

Nettesheim et al.[157] investigated the effect of ozonized gasoline on tumor production in hamsters. The ozone concentration was 2353 μg/m^3 (1.2 ppm) in excess of that which reacted with the gasoline. The resulting hydrocarbon mixture was 40 to 45 ppm methane equivalents. Animals were exposed 6 hr/day, 5 days/week for life to ozonized gasoline, to ferric oxide (Fe_2O_3), or to both. Some of the groups also received injections of the carcinogen, diethylnitrosamine (DEN). The tumorigenicity of DEN was enhanced by Fe_2O_3, but not by the ozonized gasoline. The ozonized gasoline, alone or in combination with Fe_2O_3, did not appear to be carcinogenic. When the data for the groups of ozonized gasoline plus DEN and ozonized gasoline plus Fe_2O_3 were compared with data for DEN and Fe_2O_3 control groups, and were analyzed under the assumption that the tumors of the bronchi and lungs were lethal, there was a reduction ($p < 0.05$) in tumor incidence in the groups receiving ozonized gasoline.

PULMONARY FUNCTION

Scheel et al.[181] exposed 75 rats to 3920 $\mu g/m^3$ (2 ppm) ozone for 3 hr and then measured their oxygen uptake, tidal volume, and frequency of breathing. The rats were examined at intervals over a period of 960 hr after exposure. The lungs were excised and weighed before and after drying. It was found that the water content of the lungs increased during the post-exposure period, the increase reaching a maximum after 12 hr. These authors expressed pulmonary edema in terms of water in the lung per kilogram of body weight instead of per unit of dry-lung weight. Since no data are given on body weight, the changes in lung-water content must be interpreted with caution. A decrease in minute ventilation (the volume of air breathed per minute), tidal volume (volume per breath), and oxygen uptake occurred immediately after exposure and reached minimum recorded values 8 hr after exposure. At 20 hr after exposure, all measurements had returned to their initial values. The early fall in minute ventilation and oxygen uptake could have reflected a decrease in activity (metabolic rate) secondary to acute injury of tissue and the development of pulmonary edema. The simultaneous increase in minute ventilation and decrease in oxygen consumption after 40 hr suggests that delayed impairment may have occurred.

Murphy et al.[146] exposed guinea pigs to 666 to 2646 $\mu g/m^3$ (0.34 to 1.35 ppm) ozone for a period of 2 hr. Measurements of respiratory function were made before, during, and after exposure. It was observed that respiratory rates increased while tidal volumes decreased during exposure to all concentrations. The maximum changes were different ($p < 0.05$) from pre-exposure control values for each concentration. Generally, as the concentration of ozone was increased, the magnitude of the response increased, and the response occurred sooner. After a maximum response was reached, the effects tended to remain constant for the rest of the exposure period. Values tended to return to pre-exposure control levels when the animals were returned to clean air. In a later study by Easton and Murphy,[154] guinea pigs were exposed to 9800 to 13,720 $\mu g/m^3$ (5 to 7 ppm) ozone for 2 hr. Measurements of flow resistance, frequency of breathing, tidal volume, and lung compliance were made before, during, and after exposure. A threefold increase in flow resistance occurred during exposure and reached a maximum after 1 hr, a condition that may reflect narrowing of airways (predominantly in the region of large-to-medium-sized bronchi) or smaller lung volumes. There was a 50-percent increase in frequency of breathing and a small decrease in tidal volume. Lung compliance (a measurement of distensibility) was reduced 50 percent. All measurements returned to pre-exposure levels within 60 to 90 min after termination of exposure.

In another investigation,[212] the right lung of rabbits was exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 3 hr. The left deflated lung was used as a control after the air pressure was returned to normal. The exposed lungs of rabbits sacrificed 1 and 3 days after exposure had reduced vital capacities. Those studied 7 days post exposure were not significantly reduced. Yokoyama[211] studied the ventilatory functions of guinea pigs before, during, and after a 2-hr exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone. The following results were noted: Air flow resistance increased by 113 percent ($p < 0.05$), frequency of respiration increased ($p < 0.05$), and tidal volumes decreased. Yokoyama[214] also investigated the pulmonary function of rabbits exposed to 1960 or 3920 $\mu g/m^3$ (1 or 2 ppm) for 6 hr/day for 3 to 4 days. At the higher ozone concentrations, there were increases in residual volume/total lung capacity, functional residual capacity/total lung capacity, total lung resistance, and a decrease in chest wall resistance. For 1960 $\mu g/m^3$ (1 ppm), the changes were similar, but fewer than at the higher concentration. At 1960 $\mu g/m^3$ (1 ppm), the flow volume curve was not different from control.

When cats were exposed to 510, 980, and 1960 $\mu g/m^3$ (0.26, 0.5 and 1 ppm) ozone for 2 to 6.5 hr, pulmonary flow resistance increased with increasing ozone levels. The lower ozone concentration produced alterations in fewer animals. As the length of the exposure increased from 0 to 240 min, there was a trend toward an exponential increase in resistance. Dynamic compliance (C_{dyn}) and diffusing capacity for carbon monoxide (DL_{CO}) (a measure of gaseous diffusion across the alveoli into the blood) decreased less frequently and less markedly than the alteration in pulmonary resistance. Greatest reductions in C_{dyn} and DL_{CO} were observed at the higher ozone concentration (1960 μg/m^3, or 1 ppm). There were no changes in vital capacity. Alveolar stability was also unchanged, as illustrated by the shapes of the volume-pressure curves during deflation for exposed and control animals. Pathological reports on similarly treated animals indicated severe injury to the bronchial and bronchiolar epithelium, Type 1 alveolar cells, and interalveolar capillaries. This probably contributed to the depression of diffusing capacity. No evidence of intra-alveolar edema was seen. The authors suggest that acute ozone exposure affected airway caliber more than either the transfer of CO across the alveolar-capillary membrane or alveolar surface forces.[204]

Bartlett et al.[9] exposed young rats (3 to 4 weeks old) continuously for 30 days to 392 μg/m^3 (0.2 ppm) ozone. No changes in respiratory frequency occurred. Morphometric analysis indicated that the numbers of alveoli were unchanged, but the average lung volume increased by 16 percent ($p < 0.02$) following ozone exposure. The increases ($p < 0.05$) in mean cord length and alveolar surface area of ozone-exposed animals reflect overdistension of the lung. Overdistension is also illustrated by the lower ($p < 0.05$) transpulmonary pressure that occurred in both air- and saline-filled lungs, which indicated that a decrease in tissue elasticity rather than alterations of surface tension, was responsible for the results. Using light microscopy, no morphological changes were evident. At higher concentrations of ozone, investigators[216] found no significant changes of the pulmonary pressure/volume relationship or the ratio of collagen to elastin in the lungs of rats exposed to 880 μg/m^3 (0.45 ppm) ozone for 6 hr/day for 6 to 7 weeks.

Exposure to 1960 μg/m^3 (1 ppm) ozone for 3 hr, using the unilateral lung technique, in which only one lung is exposed to ozone, resulted in a loss of elasticity in rabbit lungs immediately after exposure ($p < 0.05$) and 3 days later ($p < 0.05$).[213] By day 14, the lung had recovered, suggesting that edema, present in the exposed lung on days 1 through 3 ($p < 0.05$), could have been responsible.

EDEMAGENESIS AND TOLERANCE

Data from many sources reviewed by Coffin and Gardner[37] indicate that brief exposure to ozone may elicit edema and an acute inflammatory response in the lungs of many species. It is striking at higher levels of exposure and diminishes with descending concentrations of the gas. Thus no evidence of edema was noted in mice exposed to 1960 μg/m^3 (1 ppm) ozone for 4 hr, whereas exposure to this concentration for 2 hr resulted in very slight edema when a gravimetric measurement of lung water was employed.[181,194] These investigators[16,194] detected evidence of edema in rats after exposure to 3920 μg/m^3 (2 ppm) ozone for 3 hr. Exposures at the high level of 6272 μg/m^3 (3.2 ppm) ozone quickly produced gross evidence of edema in mice.

It is generally believed that gross edema is probably not elicited in any species exposed to ambient concentrations of ozone. More refined methods for the detection of possible edema were developed by Alpert et al.,[5] greatly increasing the sensitivity of the measurement. They injected ^{132}I-serum albumin into rats and recovered pulmonary fluid 6 hr later by lung lavage, which was then tested for radioactivity. Radioactivity in the fluid would be indicative of fluid flux across pulmonary membrane barriers, and an increase over controls would represent possible edema. After 6 hr of exposure to 490 μg/m^3 (0.25 ppm) ozone, there was no significant effect; but to 980 μg/m^3 (0.5 ppm), there was increased recovery of ^{132}I (two times that of controls) ($p < 0.001$). Simultaneous studies using lung wet weight/dry weight ratios showed an effect only with 4900 μg/m^3 (2.5 ppm) ozone ($p < 0.01$).

Another report[102] indicates that a 5-hr exposure to lower levels of ozone caused increased permeability of the alveolar-capillary membrane in mice as measured by recovery of radiolabeled albumin in the lavage fluid. However, because the objective of the study was to measure the ozone hazard in UV isolation units, the mice were exposed to varying concentrations of ozone (an average of 196 μg/m^3, or 0.1 ppm) under conditions that could be called uncontrolled relative to typical studies of ozone toxicology.

Frank et al.[69] exposed the right lung of rabbits to 4312 and 23,716 μg/m^3 (2.2 to 12.1 ppm) ozone for 3 hr, the left lung having been collapsed before exposure. When edema occurred in the right lung, changes in surfactant (a substance that lowers surface tension in the lung, helping to make normal breathing possible) behavior were observed in the left lung of some animals. No such changes were observed in the absence of edema in the right lung. These results suggest that ozone is not only capable of inducing chemical changes in exposed lungs, but also that the products of such changes are capable of producing deleterious effects or compensatory responses in nonexposed lungs.

Ozone has been found to induce tolerance, which can be described as the acquired capacity of a pretreated host to exhibit a lesser response to a challenge than would be observed in a comparable but nontreated host. Using the radiolabeled albumin technique, Alpert et al.[5] examined the role of methylprednisoline (a corticosteroid with potent anti-inflammatory properties) on edema and tolerance to edema in rats. They found that it increased susceptibility to edema production following exposure to low levels of ozone. However, when animals received the steroid before exposure to 490 $\mu g/m^3$ (0.25 ppm) ozone, they became tolerant to a subsequent ozone challenge with 1960 $\mu g/m^3$ (1 ppm) 48 hr later (i.e., edema was not produced). Untreated rats did not become tolerant.

Other work was undertaken to ascertain the mechanisms and extensiveness of tolerance. In one such study, the left lungs of Flemish Giant rabbits were catheterized and exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone for 3 hr. The right lung inspired clean air. This was followed by an 18-hr latent period, after which the whole animal was challenged with 43,120 $\mu g/m^3$ (22 ppm) for 3 hr. The pre-exposed left lung exhibited tolerance to pulmonary edema ($p < 0.001$), and the right lung did not, indicating that tolerance is a localized, rather than a systemic, effect.[4]

Similar methods were used by Gardner et al.,[78] who pre-exposed the left lung to 980 or 1960 $\mu g/m^3$ (0.5 or 1 ppm) ozone for 3 hr, and 18 hr later exposed the whole animal to either 5880 $\mu g/m^3$ (3 ppm) or 43,120 $\mu g/m^3$ (22 ppm) ozone for 3 hr. The right and left lungs were then examined separately. Again, it was found that pulmonary edema was prevented only in the pre-exposed lung ($p < 0.001$). Also, the 980 $\mu g/m^3$ (0.5 ppm) ozone produced greater tolerance than did the 1960 $\mu g/m^3$ (1 ppm) ozone pre-exposure. The same authors had demonstrated that ozone exposures resulted in an influx of polymorphonuclear leukocytes (PMN) into the lung, while the total number of alveolar macrophages remained the same. Therefore, the effect of ozone pre-exposure on this parameter was studied. The tolerant pre-exposed lung had the same total number of alveolar macrophages as the nontolerant lung, as well as a higher number of PMN. This demonstrated that the prior exposure induced further chemotaxis of PMN but had no effect on alveolar macrophages. Macrophage enzyme activities (lysozyme and β-glucuronidase) were reduced in response to ozone, and pre-exposure failed to influence the action of ozone on these enzymes. These investigations indicated that the extent of tolerance depends on the biological endpoint and is more effective against pulmonary edema than against other health effects.

Using the infectivity model, which measures the accumulated mortality of mice exposed to pollutants and aerosols of *Streptococcus pyogenes,* Gardner and Graham[77] investigated ozone tolerance. Some animals received two 3-hr exposures to the same concentration of ozone, with a 24-hr interval between the exposures. Other mice received only one 3-hr exposure to ozone. The bacterial aerosol was administered immediately after the last ozone exposure. At 196 $\mu g/m^3$ (0.1 ppm), there was only a slight difference in mortality between the mice receiving 2 and 1 exposures to ozone. As the concentration was increased, up to 1960 $\mu g/m^3$ (1 ppm), there was greater protection in those animals pre-exposed (i.e., the mortality was less in those mice receiving ozone twice, as compared to animals exposed once). However, excess mortality occurred irrespective of the number of ozone exposures, indicating that complete tolerance was not evident. The partial tolerance to infectivity that occurred at higher ozone concentrations was probably due to inhibition of edema, and not to tolerance to an ozone effect on alveolar macrophages. That ozone-induced damage to alveolar macrophages cannot be completely prevented by prior ozone exposure has been demonstrated by Gardner et al.[93]

Evans et al.[59,60] measured tolerance by studying the kinetics of alveolar cell division in rats during a period of exposure to an elevated ozone concentration (980 or 1372 $\mu g/m^3$, or 0.5 or 0.7 ppm, up to 4 days) that followed initial exposure at a lower concentration (686 $\mu g/m^3$, or 0.35 ppm for 4 days). Tolerance in this case was the ability of Type 1 cells to withstand re-exposure, and any increase in the numbers of Type 2 cells would indicate a lack of tolerance. These studies showed that tolerance to the initial concentration of ozone did not ensure total protection against re-exposure to higher concentrations (see section on morphology studies for more complete discussion).

Note that when mice were exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 1 hr and then X-irradiated with 800 R 10 days later, 60 percent of the animals survived, whereas all previously non-ozone-exposed mice died. This tolerance lasted 30 days or more.[99]

The mechanism for development of tolerance to ozone-induced edema is not well understood, but the thymus may play a role. In a study by Gregory et al.,[93] male mice thymectomized at birth were unable to develop tolerance to a 2-hr exposure of 39,200 $\mu g/m^3$ (20 ppm) ozone when they had been pre-exposed to 588 $\mu g/m^3$ (0.3 ppm) of ozone for 1 hr ($p < 0.01$).[93] Sham-operated animals exhibited tolerance under the same oxidant conditions. Contrary to these studies, Thompson[198] found that thymectomizing the animals had no effect on tolerance.

Biochemical responses in lungs of ozone-tolerant rats were studied by Chow[27] and Chow et al.[29] (see section on sulfhydryl compounds and pyridine nucleotides, and earlier section on mitochondrial enzyme activities for complete description). The lungs of tolerant animals showed relatively less injury and maintained significantly higher activities of glutathione peroxidase, glutathione reductase, and glucose-6-phosphate dehydrogenase, and higher levels of reduced glutathione. The author speculates that this higher activity in the lungs of tolerant animals may render them more resistant to peroxidation damage induced by ozone. However, with respect to the mitochondrial oxidation of succinate, re-exposure after a recovery period resulted in alterations in enzymatic activity similar to those observed after initial exposures.

Dixon and Mountain[47] concluded that edemagenesis in mice exposed to ozone probably involves the release of histamine and other endogenous products such as slow-reacting substances, but they found that lung histamine content did not parallel tolerance and that tolerance was not affected by histamine antagonists or liberators. Their conclusion was that histamine per se had no function in development of tolerance to ozone.

Extrapulmonary Effects

HEMATOLOGICAL AND SERUM CHEMISTRY CHANGES

Some of the studies to be described here have also been conducted with human blood and are discussed in Chapter 9.

Brinkman et al.[20] have shown that inhalation of 392 to 490 $\mu g/m^3$ (0.20 to 0.25 ppm) ozone over periods of 30 to 60 min by mice, rats, rabbits, and man increased the rate of sphering of red cells in vitro, with the cells losing their characteristic biconcave shape more rapidly when the diluted blood is subsequently exposed to X-radiation. These changes were greatest after 1 hr and approached control values 6 hr after exposure ended.

Menzel et al.[132] investigated the effects of ozone on erythrocyte hemolysis. Rats maintained on diets either deficient in or supplemented with vitamin E were continuously exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone, and their red blood cells were subjected to dialuric acid hemolysis to estimate plasma tocopherol. After 23 days, the erythrocytes from 60 percent of the rats depleted of vitamin E and exposed to ozone showed hemolysis. This response did not occur in the vitamin-E-depleted, air-exposed control animals until 36 days. Those animals supplemented with vitamin E did not show hemolysis, regardless of exposure. These data are believed to illustrate that ozone accelerates the depletion of tocopherol reserves.

Ross et al.[177] examined erythrocytes of rabbits exposed for 4 hr to 1960 or 5880 $\mu g/m^3$ (1 or 3 ppm) ozone. There was no significant effect, immediate or delayed, on parameters that reflect oxygen delivery by the red cell; i.e., oxyhemoglobin affinity, heme-oxygen binding site interaction, and 2, 3-diphosphoglycerate concentration.

Other studies[182] were conducted in which mice breathed 1686 $\mu g/m^3$ (0.86 ppm) ozone for 8 hr/day, 5 days/week for 6 months, after which the animals were infected with *Plasmodium berghei* (a sporozoan, of the same family as the malaria organisms, that parasitizes the red blood cells of certain mammals). Up to the 6th week of exposure, there was a significant increase in resistance of the erythrocytes to acid hemolysis.[182] Other investigators observed increased resistance to hemolysis in mice exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 30 min.[138]

Ozone may not only affect the unsaturated fatty acids of cell membranes, but it may also damage acetylcholinesterase (AChE), an enzyme bound to red cell membranes. This was studied[163] after in vitro exposure of AChE derived from bovine erythrocytes. The enzymatic activity was inhibited after exposure to ≥ 588 $\mu g/m^3$ (0.3 ppm). Eglite[53] reported that whole blood cholinesterase of rats decreased by the middle of the third month of a 93-day continuous exposure to 110 $\mu g/m^3$ (0.056 ppm), but returned to normal 12 days after exposure ended. The method of ozone measurement was not specified.

Chow et al.[30] found no statistically significant changes in the level of GSH or the activities of GSH peroxidase, GSH reductase, and G-6-PD in the erythrocytes of rhesus monkeys or rats exposed to 0.980 $\mu g/m^3$ (0.5 ppm) ozone for 8 hr/day for 7 days. However, in the lungs of the rats, increases ($p < 0.05$) of GSH and these enzyme activities were observed (see earlier section on sulfhydryl compounds and pyridine nucleotides).

Leukocytes also have been investigated. High concentrations of ozone (≥ 2500 $\mu g/m^3$, or ≥ 12.8 ppm for 2 hr) can increase the neutrophil: lymphocyte ratio of circulating blood of rabbits. However, exposure to 3920 or 2940 $\mu g/m^3$ (2 or 1.5 ppm) ozone for 5 hr did not change this ratio.[17]

P'an and Jegier[164] observed serum protein changes in rabbits exposed to 784 or 1960 $\mu g/m^3$ (0.4 or 1.0 ppm) ozone for 6 hr/day, 5 days/week for 10 months. Exposure to the lower ozone concentration resulted in a small progressive decrease of serum albumin (16 percent by day 210). Gamma globulin varied within ± 10 percent of control values over the first 90 days, but increased thereafter and was 45 percent above control after 210 days of exposure to 780 $\mu g/m^3$ (0.4 ppm) ozone. At the same ozone concentration, there was a slight decrease (approximately 8 to 10 percent from controls) of α and β globulin over the first 30 days of exposure; β globulin then increased to control levels for the remainder of the experiment. However, the α globulin of the exposed animals continued to increase, reaching 78 percent above controls at 210 days of exposure. Similar types of changes were observed at the 1960 $\mu g/m^3$ (1 ppm) exposure for 190 days, at which time α and γ globulin had increased by 46 percent and 48 percent, respectively, and albumin had decreased by 11 percent. No significant changes were observed in total serum protein concentrations of either of the exposure groups.

To determine whether ozone can cause pre-emphysematous changes in the lungs of small animals by destroying serum antitryptic factors, P'an and Jegier[165] undertook to examine the sera of rabbits exposed to 784 $\mu g/m^3$ (0.4 ppm) ozone for 6 hr/day, 5 days/week for 6 months. Except for an increase in the serum trypsin inhibitor capacity on the first day of ozone exposure, no significant changes were found. A slow but steady rise in serum protein esterase was found also after rabbits were exposed to 784 $\mu g/m^3$ (0.4 ppm) for 6 hr/day, 5 days/week for 10 months.[110] For 10 months, rabbits were exposed to 784 $\mu g/m^3$ (0.4 ppm) ozone, and the concentration of serum trypsin protein esterase tripled by the end of exposure. This rise may be related to the observed thickening of small pulmonary arteries.[166]

Numerous other changes have been observed in the blood of ozone-exposed animals. Veninga's[202] work, in which rabbits were exposed for 60 min to 392 $\mu g/m^3$ (0.2 ppm) ozone, showed a small but significant drop in total blood serotonin immediately after termination of exposure. This reduction was probably due to a loss of platelet-bound amine because there was no measurable free circulating plasma serotonin. Investigation of plasma lysozyme[28] activity showed that rats continuously exposed to 1568 $\mu g/m^3$ (0.8 ppm) ozone over 29 days had increased ($p < 0.001$) enzyme activity by the third day of exposure. The lysozyme activity remained elevated on day 10 and day 29. In another study, mice exposed to 392 $\mu g/m^3$ (0.2 ppm) ozone for 2 hr exhibited an increase in serum glutamic-pyruvic transaminase (SGPT), indicating enhanced fat deposition in the liver as well as an increase in hepatic ascorbic acid. There was no change in blood catalase, a strong reducing agent.[201]

CHROMOSOMES

There have been numerous studies designed to provide information on the possible role of ozone as a mutagenic agent. A number of investigators[42,43,95,96,97,185] have used the microorganism, *Escherichia coli*, as the indicator system for ozone-induced specific modifications of the genetic material. Fetner[65] has demonstrated the capability of ozone to produce chromosome breaks in the root meristem cells of the broad bean, *Vicia faba*. The same author[66] reports a significant delay in mitosis when living neuroblasts of the grasshopper, *Chortophaga viridifaciata*, are exposed to ozone. No effect was detected until the embryos were exposed to an ozone atmosphere present in a closed system with a solution of 3500 to 4500 μg ozone/liter. These effects were reversible. The effects of ozone-treated seawater on the oyster has also indicated the presence of fragmented nuclei, indicating chromosome breaks.[122]

Zelac et al.[217] exposed female Chinese hamsters to 470 $\mu g/m^3$ (0.24 ppm) ozone for 5 hr and then examined circulating blood lymphocytes for chromosome damage. Ozone-induced chromosomal breaks ($p < 0.05$) and aberrations were still present 6 and 15 days post exposure to ozone. The authors suggest the possibility of long-term effects from these cellular alterations. Expanding these studies, Zelac et al.[218] calculated that the exposure-adjusted break frequency was 1.67×10^{-3} breaks/cells (ppm/min). When they combined ozone (392 $\mu g/m^3$, or 0.2 ppm, for 5 hr) with radiation exposure (230 rads delivered in 5 hr), the two agents simultaneously exhibited $>$ 70 percent of the total number of breaks anticipated, assuming the actions of two agents were additive.

Gooch et al.[92] studied the cytogenetic effects of ozone in three systems: Chinese hamster bone marrow, mouse peripheral leukocytes and mouse primary spermatocytes. The hamsters were exposed to 451 $\mu g/m^3$ (0.23 ppm) for 5 hr or 10,192 $\mu g/m^3$ (5.2 ppm) for 6 hr. The mice were exposed to 294 $\mu g/m^3$ (0.15 ppm) and 412 $\mu g/m^3$ (0.21 ppm) for 5 hr, or 1940 $\mu g/m^3$ (0.99 ppm) for 2 hr. The data tended to disagree with those of Zelac et al.[217,218] in that no appreciable increase in frequency of ozone-induced chromosome-type aberrations was observed. Other researchers[179] exposed cell cultures of embryonic chick fibroblasts (total flask vol. of 160 ml) to 10 to 100 μl ozone/ml for 30 min. These cells exhibited major but nonspecific alterations in both interphase and mitotic cells at the margins and top layers of the cultures. Several hours post exposure, abnormal bi- and poly-nucleated reconstructed cells were present. A few cells had chromosome bridges in anaphase and telophase together with nuclear fragments that resembled X-ray-induced damage. Fetner[67] exposed human cell cultures in vitro to ozone (8.0 ppm by weight of ozone in O_2 for 5 and 10 min). This investigation demonstrated that ozone is capable of producing chromatid breakage in the KB human cell line.

A review article by Veninga,[202] which includes some of his own research, presents evidence for the radiomimetic properties of ozone. C-57 black mice were treated with 392 $\mu g/m^3$ (0.2 ppm) ozone for 7 hr/day, 5 days/week during gestation and then for the first 3 weeks of life. Unlimited incisor growth was found in 0.9 percent of the normal newborn mice, whereas ozone-exposed animals had an incidence of 5.4 percent. In the same species, neonatal death rose from 9 to 34 percent after exposure. In addition, blepharophimosis in another strain of normal mice occurred in 0.6 percent of control animals and in 9.6 percent of the ozone groups. Similar but less dramatic results were seen in C-57 black mice in which the rate of blepharophimosis rose from 4.5 to 9.2 percent. In another study by the same researcher,[201] binucleated lymphocytes doubled in number in murine blood after exposure to 392 $\mu g/m^3$ (0.2 ppm) for 2 hr ($p = 0.005$). The action of ozone in changing the adsorption spectra of nucleic acids[33] and in altering the pyrimidine bases of *E. coli* nucleic acids[168] may be an indication of a chromosomal effect.

CENTRAL NERVOUS SYSTEMS AND BEHAVIORAL EFFECTS

Several studies have shown tne effects of ozone on behavior patterns in animals.

Various responses of mice exposed for 30 min to various concentrations of ozone from 1176 to 16,660 $\mu g/m^3$ (0.6 to 8.5 ppm) were studied.[167] At 1176 $\mu g/m^3$ (0.6 ppm), mice began attempting to move away from the ozone. This avoidance response was more definite at 2156 $\mu g/m^3$ (1.1 ppm) ozone, and it became more pronounced as the ozone concentration increased. In addition, as ozone concentration increased, activity decreased. After a 20-min exposure to 2156 $\mu g/m^3$ (1.1 ppm), the animals appeared confused. Other researchers[112,113] exposed rats to 98, 196, 392, 980, and 1960 $\mu g/m^3$ (0.05, 0.1, 0.2, 0.5, and 1 ppm) ozone and found that the greater the concentration of gas, the greater the depression in gross motor activity ($p < 0.05$). Although not specified in the paper, it would appear that a significant response did not occur at the lowest concentration tested. In addition, the pollutant-exposed animals had prolonged periods of inactivity. The precise time of the ozone exposures that were related to the observations was not given, but it appeared to be less than 45 min. In another series of experiments,[172] subhuman primates exhibited an elevation of both simple and choice reaction time in atmospheres of 980 $\mu g/m^3$ (0.5 ppm) ozone after only a 30-min pre-exposure to this concentration. When Fletcher and Tappel[68] continuously exposed rats to 1960 $\mu g/m^3$ (1 ppm) for 7 days to ozone, they found an 84-percent reduction in voluntary activity ($p < 0.001$). Rats exposed to 110 $\mu g/m^3$ (0.056 ppm) ozone for 93 days exhibited no change in behavior or chronaxial muscle ratios.[53]

Xintaris et al.[210] found a depression of the evoked response to flash in the specific visual cortex and in the superior colliculus of rats in reponse to exposures of 980 to 1960 $\mu g/m^3$ (0.5 to 1 ppm) ozone for 1 hr. He suggested that such changes reflect a possible morphological or functional change in the central nervous system. Bokina et al.[19] reported on animals (species not identified) that were initially exposed to (time not given) 3920 to 5880 $\mu g/m^3$ (2 to 3 ppm) ozone and then subsequently exposed (time not given) to 30 $\mu g/m^3$ (0.015 ppm). When this exposure was combined with a rhythmic, light stimulus, paroxysmal activity in the olfactory analyzer structures was provoked. Evoked potential of the optic cortex was also investigated[19] during a 1.5-month continuous exposure to 50 $\mu g/m^3$ (0.026 ppm) ozone. The following observations were made: A decrease in the amplitude of the primary response of the evoked potential, a slow negative wave, and a decrease in the duration of the slow negative wave. The authors suggest that this represents a deterioration of the cortical processes. The method of ozone monitoring was not given.

EXTRAPULMONARY MORPHOLOGY

Atwall and Wilson,[7] using light and electron microscopy, found histological and ultrastructural changes in the parathyroid glands of rabbits after 4- to 8-hr exposures to 1470 $\mu g/m^3$ (0.75 ppm) ozone. The major changes found in exposed animals were large numbers of secretion granules, hyperplasia of the chief cells, proliferation and hypertrophy of the rough endoplasmic reticulum, free ribosomes and mitochondria, lipid bodies, and an accumulaton of secretion granules of the chief cells inside the vascular endothelium of the blood stream. Alterations were observed 18 and 22 hr after exposure, but by 66 hr, the cells had normal appearances. The authors hypothesize that this damage could lead to alteration of parathormone production and storage.

Brinkman et al.[20] exposed adult mice to 390 $\mu g/m^3$ (0.2 ppm) ozone 5 hr/day for 3 weeks. Structural changes in the cell membranes and nuclei of myocardial muscle fibers were produced that were reversible about 1 month following exposure. The physiological implications of these changes were not discussed.

MISCELLANEOUS EFFECTS

Gardner et al.[78] exposed mice to 1960 $\mu g/m^3$ (1 ppm) ozone for 3 hr/day up to 7 days. Immediately after exposure, the animals were injected with sodium pentobarbital and the induction time for sleep and the actual sleeping time were determined. The induction time was not affected by ozone; however, sleeping time was altered. After the second ozone exposure, there was a 13-min prolongation in sleeping time ($p < 0.05$), whereas the increase was 9.2 min ($p < 0.05$) after 3 days of ozone treatment. No significant changes were evident after a single exposure or after four or more successive daily exposures. After seven daily exposures to 1960 $\mu g/m^3$ (1 ppm) ozone, the concentration was increased to 9800 $\mu g/m^3$ (5 ppm) for 3 hr. This increased the sleeping time by 59.5 min ($p < 0.001$) and was taken to indicate that tolerance did not occur. The authors hypothesized that these effects may reflect alterations in cytochrome P_{450} (cyt. P_{450}), a microsomal enzyme (a mixed function oxidase) that oxidizes pentobarbital. Goldstein and Balchum[83] investigated the possible role of this enzyme in ozone-induced toxicity. They found that injections of phenobarbital (an inducer of cyt. P_{450}) decreased ($p = 0.05$) the survival times of rats exposed to a lethal ozone concentration (157,680 $\mu g/m^3$, or 8 ppm), whereas treatment with allylisopropylaceta-mide (which destroys cyt. P_{450}) increased the survival time of ozone-exposed animals. Goldstein et al.[88] also showed that there was a decrease ($p < 0.01$) in lung microsomal cyt. P_{450} levels immediately, or 1, 2, 3, or 5 days after rabbits were exposed to 1960 $\mu g/m^3$ (1 ppm) ozone for 90 min. (See earlier section on other biochemical alterations.) Liver cyt. P_{450} levels of the ozone-exposed rabbits were not significantly different from control immediately or 1 day post exposure.

The effects of ozone on pulmonary arterial pressure were investigated in 31 dogs exposed to 1960 $\mu g/m^3$ (1 ppm) for 17 months for varying hours each day.[14] Three dogs developed pulmonary arterial hypertension, and 9 dogs had excessive systolic pressure. Because there was no proportional relationship between pulmonary arterial hypertension and oxidant exposure, it was suggested that the results seen were due to genetic susceptibility.

Roth and Tansy[178] exposed rats to 490 $\mu g/m^3$ (0.25 ppm) ozone for 2 hr and found no differences in gastric secreto-motor activities, although a temporary effect was present at higher levels.

Trams et al.[199] found no lipid peroxidation in brain tissue of dogs chronically (8 to 24 hr/day) exposed to 1960 $\mu g/m^3$ (1ppm) ozone for 18 months. Monoamine oxidase activity was increased ($p < 0.05$) in animals exposed for 8 to 16 hr/day, but it was decreased in those dogs exposed for 24/hr day. Catechol-o-methyltransferase activity was decreased ($p < 0.05$) for all periods of exposure. There were no significant changes in 5'-nucleotidase, acetylcholinesterase, and ATPase activities or in the levels of catecholamines.

The functional state of the adrenal cortex was investigated by Eglite.[53] Rats were exposed to 110 $\mu g/m^3$ (0.056 ppm) continuously for 93 days. After 6 weeks, the levels of urinary 17-ketosteroids were elevated and remained elevated during the course of the experiment. The ascorbic acid content of the adrenal decreased during exposure, but returned to normal levels 15 days after ozone exposure ceased. The method of ozone measurement was not specified.

Quantitative analyses of urinary constituents of rats exposed to ozone ranging from 1568 to 2940 $\mu g/m^3$ (0.8 to 1.5 ppm) 5 days/week for 18 weeks were made by Hathaway and Terrill.[98] The urine of the exposed animals had lower ($p<0.05$) titratable acidity on days 91, 98, and 112, and a higher ($p<0.05$) pH on days 98 and 112, but not day 91. On various days (days 3 to 84), there were no significant changes in urinary creatinine, creatine, uric acid/creatinine, and amino acid nitrogen/creatinine. For the first 53 days of the study, caging arrangements partially hampered the animals' ability to obtain food, and this may have influenced the results.

There are suggestive data that exposure to ozone may accelerate the aging process. Bjorksten and Andrews[13] and Bjorksten[12] have presented the concept that aging may be due to irreversible cross-linking between macromolecules, principally proteins and nucleic acids. They included aldehydes in their list of active cross-linking agents. Aldehydes are potential cross-linking agents and may be produced in the lung by ozone exposure.

Stokinger[194] reported accelerated or premature aging in rabbits after 1 year of weekly 1-hr exposures to ozone (no concentration given). His evidence included premature calcification of the sternocostal cartilage, appearance and coarseness of the pelage (hairy system of the body), severe depletion of body fat, and general signs of senescence, such as dull corneas and sagging conjunctivae. It has been suggested that the radiomimetic properties of ozone are implicated in the effects on aging.

Summary and Conclusions

Animal toxicological studies in conjunction with human clinical and epidemiological studies serve to provide information on the health effects of ozone. Investigations with animals are valuable in that they both support and extend the information derived from other experimental approaches. For example, with animals it is possible to use long-term exposures, controlled pollutant mixtures, and invasive measurement techniques that cannot be employed with humans because of ethical considerations. A major area of concern in animal studies involves the validity of extrapolating the results to man. Since many physiological mechanisms are common to animals and man, it can be hypothesized that if ozone causes a particular health effect in several animal species, it is likely to cause similar effects in exposed humans. But at this time, it is not possible to predict the concentration of ozone that would be responsible.

Interpretation of animal study results regarding the severity of the effect and its implication to human health is also difficult. Exposure to ozone results in an array of effects that vary from relatively small changes observed at low concentrations after short-term exposure, to gross alterations (including death) with higher concentrations and longer exposure times. A statistically significant change observed after exposure to ozone may be of unknown physiological significance. However, it could be hypothesized that any detectable deviation from normal is potentially undesirable.

With the foregoing as background, the results of the animal studies can be summarized. The weight of the data from numerous laboratories indicates that exposure to ozone results in alterations of host defense mechanisms, pulmonary morphology, and pulmonary function; biochemical changes; and effects on circulating blood cells and sera. In mice, a 3-hr exposure to concentrations of ozone as low as 157 $\mu g/m^3$ (0.08 ppm) results in a concentration-related increase in susceptibility to infectious respiratory disease, as measured by an increase in mortality from laboratory-induced pneumonia. Other studies employing this model system indicated that the presence of other pollutants (nitrogen dioxide and sulfuric acid) would have additive effects with ozone. Investigations of host defense mechanisms have indicated that a 2.5- to 4-hr exposure of ozone depresses pulmonary bactericidal activity (1176 $\mu g/m^3$, or 0.6 ppm) and causes increased lability of functional and biochemical alterations of alveolar macrophages (196 to 980 $\mu g/m^3$, or 0.1 to 0.5 ppm), which are primarily responsible for maintaining sterility of the deep lung.

Numerous investigations in a variety of animal species have shown that exposure to 196 to 1568 $\mu g/m^3$ (0.1 to 0.8 ppm) ozone from a few hours to several days alters the activity of several enzymes and other biochemical constituents (proteins and lipids) in the lungs. At the 196-μg ozone/m^3 (0.1-ppm) concentration, a 7-day continuous exposure of rats maintained on a vitamin E level typical of that in the standard American diet caused an increase in oxygen consumption of pulmonary tissue. A similar effect was not noted in vitamin-E-supplemented rats until the concentration was increased to 392 $\mu g/m^3$ (0.2 ppm). Additional research has also shown that vitamin E can protect the host from the adverse effects of ozone. In a comparative study, rats were affected (increased O_2 consumption of pulmonary tissue) by a lower concentration (392 $\mu g/m^3$, or 0.2 ppm) of ozone than monkeys (686 $\mu g/m^3$, or 0.35 ppm) following a 7-day intermittent exposure. The activities of other enzymes (e.g., glucose-6-phosphate dehydrogenase, glutathione reductase, and glutathione peroxidase) and concentrations of glutathione also increase following ozone exposure to levels equal to or greater than 392 $\mu g/m^3$ (0.2 ppm) for several days (typical experiments were for 7 days, but in some studies, effects were noted earlier). Since these enzyme systems can detoxify oxidizing substances such as ozone and its reaction products, these changes are interpreted as representing an increased protection against ozone. Thus the increased enzymatic activity might represent a reaction to potential toxicity, rather than a toxic response itself.

Many of the biochemical effects of ozone have been attributed to concomitant morphological alterations of the lung. Concentrations of ozone equal to or greater than 392 $\mu g/m^3$ (0.2 ppm) can lead to an increase in the number of Type 2 cells that replace Type 1 cells in both rats and monkeys. Since Type 2 cells are rich in mitochondria and have higher metabolic activity, this situation would lead to increased activity of several enzyme systems in the lung. In monkeys, the predominant injury is located at the respiratory bronchiole (8 hr/day for 7 days). With increasing concentrations of ozone, the extent of the damage expands distally. In rats, which do not have respiratory bronchioles, the principal site of damage was the alveolar duct. The lesion generally reaches a peak in 3 to 5 days (after continuous or intermittent exposure), and then progressively diminishes, even during continuing ozone exposure. The meaning of these changes is not immediately apparent, but it has been suggested that the alterations are part of an adaptive response. Morphological alterations caused by ozone can be quite extensive and involve areas of the lung other than the area of the respiratory bronchiole and alveolar duct. For example, a 3-month continuous exposure to 1725 $\mu g/m^3$ (0.88 ppm) can produce emphysema-like structures in rats. Intermittent exposure (6 hr/day, 5 days/week for 10 months) to 984 $\mu g/m^3$ (0.4 ppm) can produce emphysematous-like lesions in rabbits.

Alterations in pulmonary function have been observed in animals following short-term exposures equal to or greater than 510 $\mu g/m^3$ (0.26 ppm) ozone. Pulmonary flow resistance (a measure of airway diameter) and respiratory rates increased, and tidal volume decreased as the concentration increased. At a higher concentration (1960 $\mu g/m^3$, or 1 ppm), compliance (a measure of lung distensibility) and carbon-monoxide-diffusing capacity (a measure of alveolar gas exchange) decreased. Young rats continuously exposed (30 days) to 392 $\mu g/m^3$ (0.2 ppm) were also affected, as indicated by increased lung volumes and other changes that were interpreted as a decrease in lung tissue elasticity. Sensitive measurements of small airway disease, such as those made in humans, are very difficult to perform in small animals because of the size and voluntary maneuvers of the subject that are required. Therefore it would be expected that many of the slight-to-moderate morphological alterations typically seen in animals would not be detected by pulmonary function measurements.

The health effects of ozone are affected by other environmental factors such as dietary levels of vitamin E and the presence of other pollutants that have already been mentioned. The potential of ozone to modify the transformation of other environmental contaminants or drugs is suggested by the results of several studies that show that 1.5- to 3-hr exposures to 1470 to 1960 $\mu g/m^3$ (0.75 to 1.0 ppm) ozone decrease the concentration of mixed-function oxidases of pulmonary tissue. In another experiment, a 2-day exposure (3 hr/day) to 1960 $\mu g/m^3$ (1 ppm) ozone was interpreted as having a similar effect, perhaps on the liver. Lower concentrations were not tested. Nonetheless, such data would lead to the inference that by lowering the concentration of mixed-function oxidases that either detoxify or activate chemical compounds (depending on the compound), ozone would influence the toxicity of other environmental chemicals that are substrates for this enzyme system. The net result depends on whether the toxicity of the chemical would be enhanced or decreased by mixed-function oxidase activity.

A considerable number of research studies have addressed tolerance to ozone, a phenomenon by which a prior dose of ozone will protect an animal from a later higher (or equivalent) dose of ozone. Though such work is interesting, its relevance to the protection of health is difficult to assess, since concentrations of ozone found to induce tolerance also elicit alterations in biological endpoints. In addition, tolerance is only highly efficient against edema and results in partial or no protection against some of the alterations in pulmonary biochemistry, morphology, and host defense mechanisms against infectious pulmonary disease.

In some studies of host-defense mechanisms, biochemistry, and morphology, concentration-response relationships were investigated. As the concentration of ozone was increased, the effect also increased. Also, in most cases in which different exposure modes were compared, there were no significant differences between intermittent (7 to 8 hr/day) and continuous exposure. From such information, it can be postulated that insufficient recovery occurs during the clean-air period of the intermittent exposure mode.

The persistence of the health effects caused by ozone has only been measured in a few studies and varies considerably. Some pulmonary biochemical alterations exhibit a plateau or continue to increase during exposure and then subside immediately or a few days following cessation of exposure. Other enzyme activities remain elevated a few weeks after exposure. The typical morphological lesion (in which Type 2 cells replace Type 1 cells) reverts toward normal during ozone exposure. However, more severe pathological effects such as emphysema would remain throughout the life of the animal.

Though most ozone toxicological studies have focused on the lung, systemic effects have also been observed. Extrapulmonary effects might be a result of neurohormonal mechanisms initiated by ozone or of the toxic action of the reaction products of ozone affecting blood components as they pass through the capillaries surrounding the alveoli; or these reaction products could possibly enter the circulation, exerting effects at distant target sites. Also conceivable is that ozone itself could cross the alveoli and affect blood components, though such an event is unlikely, since mathematical predictions estimate that only a miniscule dose would reach the level of the alveoli. Whatever the reason(s), extrapulmonary effects have been noted at concentrations as low as 392 $\mu g/m^3$ (0.2 ppm) for a 30-min exposure.

Numerous alterations in erythrocytes and in the sera have been observed following ozone exposure. Increased lysis of red blood cells of exposed (980 $\mu g/m^3$, or 0.5 ppm for 23 days, continuous) vitamin-E-deficient animals has been reported. An enzyme bound to red cell membranes is also affected by ozone. Serum protein changes have also been shown to occur in animals exposed to ozone levels between 392 and 1568 $\mu g/m^3$ (0.2 and 0.8 ppm). Levels of albumin, globulins, and serum enzymes were altered after a 6- to 10-month intermittent exposure to 784 $\mu g/m^3$ (0.4 ppm). Exposure to 392 $\mu g/m^3$ (0.2 ppm) for 1 hr reduced total blood serotonin, and a 3-week intermittent exposure to this ozone level increased serum glutamine pyruvic transaminase.

The influence of ozone on chromosomes has also been investigated and is the subject of some controversy. In one study, Chinese hamsters exposed to 392 $\mu g/m^3$ (0.2 ppm) ozone for 5 hr exhibited an increased number of chromosomal breaks in circulating lymphocytes, which were detectable up to 15 days following the end of the ozone exposure. Another investigation using approximately the same ozone exposure failed to demonstrate any chromosomal aberrations in Chinese hamster bone marrow, mouse peripheral leukocytes, and mouse primary spermatocytes. A number of investigations have shown that bacteria, plant cells, oyster cells, and chick fibroblasts exposed to ozone in vitro exhibited chromosomal alterations. Because of these conflicting data, no definitive conclusions can be drawn at present regarding the effects of ozone on chromosomes. Even if the work with circulating lymphocytes were replicated in the future, the biological significance of the findings (beyond that of indicating systemic chromosomal damage from an environmental agent) would be difficult to assess. Lymphocytes are considered to be a terminal cell; but for proper immune functioning, they must divide and form an additional line of cells. Consequently, immune functioning could be affected by ozone. However, a potential danger to human health—that of chomosomal aberrations of germ cells (i.e., ova or sperm)—has not been directly observed. In spite of the conflicting results of ozone with chromosomes, mutagenic effects have been observed. The offspring of mice exposed for 7 hr/day, 5 days/week to 392 $\mu g/m^3$ (0.2 ppm) during gestation and the first 3 weeks of life exhibited increased neonatal deaths and mutagenic effects such as increased incisor growth and blepharophimosis (a narrowing of the slit between the eyelids).

A number of other extrapulmonary effects have also been reported. Structural changes have been found in mouse heart muscle following a 3-week (5 hr/day) exposure to 392 $\mu g/m^3$ (0.2 ppm) and in the parathyroid glands of rabbits after a 4- to 8-hr exposure to 1470 $\mu g/m^3$ (0.75 ppm). Short-term (1 hr or less) exposures of greater than or equal to 980 $\mu g/m^3$ (0.5 ppm) have caused alterations in behavior and in responses of the central nervous system (i.e., decreased activity and depression of evoked reponse to flash).

Review of the literature of the health effects of ozone provides strong evidence that in animals, a large variety of pulmonary and extrapulmonary alterations are produced after a short-term exposure to concentrations between 392 and 980 $\mu g/m^3$ (0.2 and 0.5 ppm). Pulmonary biochemical alterations and an increased susceptibility to infectious disease have been observed at levels between 157 and 392 $\mu g/m^3$ (0.08 and 0.02 ppm). A prudent conclusion from the animal toxicological data is that humans exposed to ozone may experience similar effects, but it is not possible to predict precisely the severity of the effects or the concentrations at which they may occur in man.

EFFECTS OF PHOTOCHEMICAL OXIDANTS ON EXPERIMENTAL ANIMALS

Experimental Data

Investigations have been conducted of the potential biological actions of a complex photochemical reaction mixture produced by irradiating mixtures of air and auto exhaust under laboratory conditions that simulated real driving patterns and solar irradiation.[100,101,104,142,144] The effects of both nonirradiated and irradiated exhaust mixtures were studied. Clearly, irradiation of the air-exhaust mixture led to the formation of photochemical reaction products that were biologically more active. The relative proportions of the suspect biologically active chemical species varied with the total concentration of exhaust gases in the irradiated mixture and with the duration of irradiation.[142,144] Single-inhalation exposure studies lasting a few hours demonstrated that irradiation of exhaust mixtures led to greater effects on respiratory mechanics (increased flow resistance and tidal volume and decreased breathing frequency) in guinea pigs, greater reduction in voluntary running activity of mice, increased susceptibility to infection, and slightly greater carboxyhemoglobin formation in rats, compared with animals exposed to the same total concentration of exhaust gases that were not irradiated. The concentration of total oxidant as expressed by ozone in these experiments ranged between 588 and 1568 $\mu g/m^3$ (0.30 and 0.80 ppm) in the irradiated-exhaust mixtures. Only a trace or no oxidant was detected in the unirradiated exhaust. The irritant aldehydes, formaldehyde and acrolein, were also present in higher concentrations (0.39 to 2.42 ppm and 0.09 to 0.2 ppm, respectively) in the irradiated atmospheres. Formaldehyde concentrations were between 0.12 ppm and 0.38 ppm, and acrolein ranged from 0.02 to 0.07 ppm in the unirradiated exhaust chamber. The effects that were noted were reversible within a few hours when the animals returned to clean air. The effects in animals exposed to the irradiated-exhaust mixture are not necessarily uniquely characteristic of ozone, but most of them could have been produced by ozone.

Murphy et al.[142,144] found that the nature of changes in respiratory mechanics in guinea pigs exposed to irradiated exhaust varied according to the ratio of oxidant to aldehyde concentrations (formaldehyde and acrolein were measured), and this ratio in turn varied with the duration of irradiation of the air-exhaust mixture. Thus when the oxidant:aldehyde ratio was low, the guinea pig respiration pattern resembled that reported for animals exposed to irritants such as formaldehyde and acrolein[6,143] and was characterized by increased pulmonary flow resistance, increased tidal volume, and decreased frequency of breathing. Increasing the ratio resulted in a shift in the pattern of respiration toward that produced by deep-lung irritants (e.g., ozone and nitrogen dioxide[146]), namely decreased tidal volume and increased frequency, although the increased flow resistance typical of the aldehyde effect persisted. This interactive effect of an oxidant-aldehyde mixture could be reproduced by a simple mixture of ozone and acrolein.[142]

TABLE 8-1. PULMONARY EFFECTS OF OZONE: HOST DEFENSE MECHANISMS

Ozone, $\mu g/m^3$	Ozone, ppm	Length of exposure	Observed effect(s)	Species	References
1960	1	17 hr before bacteria	Decrease in bacterial pulmonary deposition; decrease in bactericidal activity.	Mice	Goldstein et al.[90]
1960	1	3 hr	Bacteria observed in the blood sooner and with increased frequency.	Mice	Coffin and Gardner[36]
1764	0.5	1-4 hr	Increased nasal, but not lung, deposition and growth of virus. Decreased minute ventilation.	Mice	Fairchild[61,62]
1568	0.8	11 days	Decreased production of interferon by tracheal epithelial cells.	Mice	Ibrahim et al.[109]
1372- 1764	0.7- 0.9	3 hr	Increased mortality of mice pre-exposed over those animals not so treated. Aerosols of *S. pyogenes* received by all animals immediately after O_3 exposure.	Mice	Coffin and Blommer[34]
1372	0.7	7 days	Deficiency of vitamin E further reduced bactericidal activity after 7 days.	Rats	Warshauer et al.[203]
1176	0.6	4 hr after bacteria	No effect on bacterial deposition and mucociliary clearance. Decreased bactericidal activity.	Mice	Goldstein et al.[89,90]
980	0.5	3 hr	Decreased enzyme activity in alveolar macrophages; increase in number of pulmonary **polymorphonuclear leukocytes** (appears to be linearly related to dose).	Rabbits	Alpert et al.[3]
980	0.5	16 hr/day × 7 months	No effect on clearance of polystyrene and iron particles.	Rabbits	Friberg et al.[73]
980	0.5	3 hr	Increased fragility of alveolar macrophages.	Rabbits	Dowell et al.[48]
980	0.5	2 hr	Decrease in agglutination of alveolar macrophage, indicating membrane alterations.	Rats	Goldstein et al.[85]
980	0.5	3 hr	Increased red blood cell rosette formation by lectin-treated alveolar macrophages.	Rabbits	Hadley et al.[94]
980	0.5	3 hr	Reduction in phagocytosis of alveolar macrophage.	Rabbits	Coffin and Gardner[36]
785	0.4	3 hr before bacteria	Lower deposition, but subsequently a higher number of bacteria present due to reproduction.	Mice	Coffin and Gardner[36]
784	0.4	4 hr	Bactericidal activity inhibited by O_3, but no role played by an induced silicotic condition.	Mice	Goldstein et al.[84]
784	0.5	17 hr before bacteria or 4 hr after bacteria	Physical clearance not affected, but bactericidal activity affected. No synergism noted with $NO\beta$.	Mice	Goldstein et al.[91]
490	0.25	3 hr	Lysozyme, acid phosphatase, and β-glucuronidase activities of alveolar macrophages reduced.	Rabbits	Hurst et al.[106]
196	0.1	2.5 hr in vivo or 30 min in vitro	Lung protective factor partially inactivated. (Appears to be dose-related.)	Rabbits	Gardner[76]
196	0.1	3 hr	Increased mortality when *S. pyogenes* aerosol challenge received immediately after exposure. Additive effect from simultaneous exposure to ≥ 3760 $\mu g/m^3$ (2 ppm) NO_2 and ≥ 98 $\mu g/m^3$ (0.05 ppm) O_3.	Mice	Ehrlich et al.[54]
157 196	0.08 0.1	3 hr	Significant increase in mortality of mice exposed to aerosols of *S. pyogenes* during ozone exposure.	Mice	Miller et al.[135]
157	0.08	3 hr	Increased mortality in mice challenged with aerosols of *S. pyogenes* immediately after exposure.	Mice	Coffin et al.[35]

TABLE 8-2. PULMONARY EFFECTS OF OZONE: BIOCHEMISTRY

Ozone, μg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
1960	1	1 hr	Carbonyl compounds found.	Rabbits	Buell et al.[23]
1960	1	Continuous	50% of vitamin-E-depleted group died in 8.2 days; 50% of vitamin-E-supplemented group died in 18.5 days.	Rats	Roehm et al.[174]
1960	1	90 min	Decreased lung microsomal cytochrome P_{450} levels after 1,2,3, or 5 days. The greater depression at 3.6 days following exposure.	Rabbits	Goldstein et al.[88]
1568	0.8	Continuous 7 days	Decreased protein synthesis, day 1; increased protein synthesis, days 2 and 3 (remained elevated and unchanged).	Rats	Mustafa et al.[149]
1568	0.8	Continuous 7 days	Increased rates of collagen and non-collagenous protein synthesis.	Rats	Hussain et al.[107]
1568	0.8	Continuous 3 days	Increase in protein synthesis, non-protein sulfhydryl content and activities of GSH peroxidase, GSH reductase and G-6-PD. Complete recovery 9 days after exposure ceased. Increased rate of mitochondrial succinate oxidation returning to normal levels 9 days after the original exposure ceased.	Rats	Chow et al.[29]
1568	0.8	Continuous 1-30 days	Increased rates of O_2 consumption, reaching a peak at day 4 and remaining at a plateau for the remainder of the 30 days. Most increase in the oxidation of succinate. Also an initial decrease (day 1) and a subsequent increase (day 2) in the activity of succinate-cytochrome-C reductase activity which plateaued between days 3 and 7.	Rats	Mustafa and Lee[150]
1568	0.8	Continuous 4-7 days	Increased rate of glucose consumption, pyruvate and lactate production after 4 days of exposure. Increased MAO activity after 7 days of exposure.	Rats	Mustafa et al.[149]
1568	0.8	7 days	Increased activities of hexose monophosphate shunt and glycolytic enzymes of lung.	Rats	Chow and Tappel[31]
1568 1960	0.8 1	10 days	Decreased cytochrome-C-reductase activity; increased G-6-PD activity; no change in sulfhydryl levels.	Rats	Delucia et al.[44,45]
1470	0.75	3 hr	Reduction in activity of benzpyrene hydroxylase in both tracheobronchial mucosa and lung parenchyma.	Hamsters	Palmer et al.[160,161]
1470	0.75	Continuous 30 days	Decreased activities of GSH peroxidase, GSH reductase, G-6-PD, 6-P-GD, and pyruvate kinase on day 1. Thereafter, increase in most of these enzyme activities until day 10, at which time, beginning of a slight decrease. At day 30, still elevated over control.	Rats	Chow and Tappel[31]
1372 1568	0.7 0.8	Continuous 7 days	Increased formation of malonaldehyde. Activities of GSH peroxidase and G-6-PD partially inhibited as a logarithmic function of dietary vitamin E. Increased activity of GSH reductase not affected by vitamin E.	Rats	Chow and Tappel[32]
1372 1568	0.7 0.8	Continuous 5 days 7 days	Increase in lysosomal hydrolases in homogenates and tissue sections. Increase in protease and peptidase seen along with other enzymatic changes. Increased activity of acid phosphatase in alveolar macrophages, terminal airway, epithelium, and adjacent structures.	Rats	Castleman et al.[25,26] Dillard et al.[46]

TABLE 8-2. PULMONARY EFFECTS OF OZONE: BIOCHEMISTRY (cont'd).

Ozone, $\mu g/m^3$	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
980 1960	0.5 1	Continuous 9 days	Alterations of lung tissue lipids. Greatest change an increase in arachidonic acid, which occurred to a greater extent in vitamin-E-deficient rats. Decreases in linolenic, oleic, stearic, and palmitic acid.	Rats	Menzel et al.[132]
980 1960	0.5 1	4 hr Continuous 1-2 weeks	Release of surfactant lecithins and decreased lecithin formation.	Rabbits	Kyei-Aboagye et al.[117] Setp et al.[186]
980 1960	0.5 1	Continuous 9 days	Vitamin E acts as an antioxidant and protects against some ozone alterations (mortality, high increases in arachidonic acid content, and lipid peroxidation).	Rats	Fletcher and Tappel[68] Roehm et al.[174,175] Shakman[188] Menzel et al.[132]
980	0.5	8 hr/day 7 days	Greater increases in the activities of GSH peroxidase, GSH reductase, and G-6-PD exhibited by rats. Only slight increases by monkeys in these enzyme activities.	Rats, monkeys	Chow et al.[30]
980 1568	0.5 0.8	8 hr/day 7 days	Increased succinate oxidase activity. Increases not significant at lower concentrations.	Monkeys	Mustafa and Lee[150]
980 1568	0.5 0.8	Continuous 7 days	Increased prolyl hydroxylase activity. No change at lower concentrations. At 1568 $\mu g/m^3$, partial return of activity to normal after a 30-day recovery, but still elevated. Increased hydroxyproline after 3 days of exposure to 1568 $\mu g/m^3$, remaining elevated after 28 days of recovery.	Rats	Hussain et al.[108]
392 686 980 1568	0.2 0.35 0.5 0.8	8 hr/day 7 days	Increased activities of GSH peroxidase, GSH reductase, G-6-PD, NADPH-cytochrome-C reductase, succinate oxidase, acid phosphatase, and B-N-acetyl-glucosaminidase. A significant correlation found between ozone concentration and increased enzyme activities.	Monkeys	Dungworth et al.[50] Mustafa and Lee[150]
392 980 1568	0.2 0.5 0.8	Continuous (7-8 days) or intermittent (8 hr/day for 7 days)	For the continuous exposure to the two higher concentrations, increased activities GSH peroxidase, GSH reductase, and G-6-PD. At the lower concentration (continuous), increased activities of GSH peroxidase and GSH reductase. A linear increase in all three enzyme activities as the concentration of ozone was increased. Increased O_2 consumption (using succinate-cytochrome-C reductase activity fairly proportional to ozone concentration. Similar results obtained for intermittent exposure groups.	Rats	Chow et al.[28] Mustafa and Lee[150]

TABLE 8-2 PULMONARY EFFECTS OF OZONE: BIOCHEMISTRY (cont'd).

Ozone, μg/m	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
392 980 1568	0.2 0.5 0.8	Continuous 7 days	Increased activities of NADPH-cytochrome-C reductase and G-6-PD.	Rats	Mustafa and Lee[150]
392 980 1568	0.2 0.5 0.8	Continuous Intermittent (8 hr/day) 7 days	Activities of G-6-PD and NADPH-cytochrome-C reductase and succinate oxidase increased in an ozone-dose-dependent fashion following exposure. No significant differences found between the intermittent and continuous exposure groups.	Rats	Schwartz et al.[184]
392 1568	0.2 0.8	Continuous 10-20 days 7 days	At the higher concentration: Increase in the lung mitochondrial ozone consumption in oxidation of 2-oxyglutarate and glycerol-1-phosphate and the number of Type 2 alveolar cells that are rich in mitochondria. At the lower concentration: Increase in O_2 consumption.	Rats	Mustafa et al.[148,187]
392 1568	0.2 0.8	8 hr/day or continuous 2-90 days	Decreased glycoprotein secretion by tracheal explants.	Rats	Last et al.[118]
392 980 1568	0.2 0.5 0.8	Continuous 7 days	Increased superoxide dismutase activity as ozone concentration increased.	Rats	Mustafa et al.[151]
392 980 1568	0.2 0.5 0.8	Continuous (8 days) intermittent (8 hr/day, 7 days)	Increased lysozyme activities at 1568 μg/m³ (continuous). No significant changes following intermittent exposure.	Rats	Chow et al.[28]
196 392	0.1 0.2	Continuous 7 days	Significant increase in succinate oxidase activity at 196 μg/m³ in those animals maintained on 11 ppm vitamin E. Increased succinate oxidase activity only at 392 μg/m³ for those rats receiving 66 ppm vitamin E.	Rats	Mustafa and Lee[150]

All the functional effects observed in short-duration experiments with laboratory-produced photochemical smog mixtures could have been due to ozone alone if one considers total oxidant concentraion of the mixture equivalent to ozone concentration. One possible exception was the increase in respiratory flow resistance in guinea pigs, which is more characteristic of an irritant aldehyde. The increase in respiratory frequency in guinea pigs is most probably due to the oxidant (or ozone) content of the mixture.

Comparison of the concentrations for equal effectiveness in decreasing the spontaneous running activity in mice also suggests that this action of the mixture may be largely due to oxidant. Likewise, the oxidant content of irradiated auto exhaust appears to explain adequately the increase in susceptibility to respiratory infection in mice exposed to the mixture. These conclusions must be qualified, since the possibility exists that other chemical species, such as free hydroxyl radicals, which were not measured, might also have produced a similar effect. Nevertheless, it is reasonable to conclude that many of the effects produced by exposures to complex photochemical oxidant mixtures are due to ozone.

Hueter et al.[104] exposed animals to irradiated automobile exhaust for periods of 6 weeks to 23 months. The concentrations were cycled to simulate daily pollution concentrations in urban cities. Daily peak concentrations of carbon monoxide of 20, 50, 60, and 100 ppm were

TABLE 8-3. PULMONARY EFFECTS OF OZONE: MORPHOLOGY

Ozone, μg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	References
1960- .510	1-0.26	4-6 hr	Loss of ciliated epithelium, damage to ciliated cells and mitochondria in some airways, swelling and desquamation of Type 1 alveolar cells, and swelling and breakage of interalveolar capillaries. Apparently dose-related.	Cats	Boatman et al.[15,16]
1960	1	8 hr/day	Damage roughly proportional to time and dose of exposure, fibrous elements deposited, lumina of small airways reduced, metaplasia of columnar and cuboidal epithelium.	Dogs	Freeman et al.[72]
1568	0.8	Continuous 3-5 months	Thickening of alveolar septa and increase in number of alveolar macrophages. Emphysematous and fibrotic changes.	Rats	Stephens et al.[192]
1058 1725	0.54 0.88	Continuous 6 months	Major site of injury at the junction of the respiratory bronchiole and the alveolar duct. A multitude of alterations observed at the lower concentration. After 3 weeks, terminal bronchioles seemed to return to normal appearance. In general, at the higher concentrations, pathological changes similar but more extensive and occurred earlier. After 3 months, emphysema-like structures seen.	Rats	Freeman et al.[70]
1176	0.6	6-7 hr/day 1-2 days	Younger mice more sensitive than older animals. Swelling of epithelial alveolar lining cells and endothelium cells. Occasional breaks in basement membrane.	Mice	Bils[11]
980 1568	0.5 0.8	8 hr/day 7 days	Lesions in centriacinar region, hyperplasia of nonciliated epithelial cells and intraluminal accumulation of macrophages. Replacement of Type 1 with Type 2 cells.	Rhesus monkeys, mice	Mellick et al.[126]
980	0.5	6 hr	Lowered rates of DNA synthesis in alveolar cells.	Aging mice	Evans et al.[59]
980 1568	0.5 0.8	2,4,6,8 or 24 hr/day for 7 days	Accumulation of inflammatory cells in centriacinar region of lung.	Rats	Brummer et al.[21]
784	0.4	6 hr/day 5 days/week 10 months	Emphysematous and vascular-type lesions observed in the lung, and small pulmonary arteries thicker.	Rabbits	P'an et al.[162]
686- 980	0.35- 0.5	Continuous 8 days	Type 1 cells replaced by Type 2 alveolar cells. No further tissue damage after 4th day.	Rats	Evans et al.[57]
588	0.3	3 hr/day 16 days	Presence of hemispheric extrusions of cilia, and small round bodies on surface of airways. Changes in Clara cells.	Rats	Sato et al.[180]
392	0.2	2 hr	Type 1 cells replaced by Type 2 alveolar cells after 24 hr.	Rats	Stephens et al.[190] Evans et al.[58,60]
392, 686 980, 1568	0.2, 0.35 0.5, 0.8	8 hr/day for 7 days	Pulmonary lesions occurring in respiratory bronchioles. Hyperplasia and hypertrophy of bronchiolar epithelium. Increase in Type 2 cells. Damage to ciliated and Clara cells. Rats and monkeys equally susceptible.	Rhesus and Bonnet monkeys, rats, mice	Mellick et al.[126] Dungworth[49] Schwartz[183] Schwartz et al.[184]

established in four sets of exposure chambers. There was considerable loss of ozone and nitrogen dioxide on chamber walls, cages, and animal fur, so the concentrations of chemically reactive gases to which the animals were actually exposed probably ranged from about 78 to 392 $\mu g/m^3$ (0.04 to 0.2 ppm) for ozone and about 280 to 940 $\mu g/m^3$ (0.15 to 0.5 ppm) for nitrogen dioxide. Interpretation of these studies is difficult because of loss of contaminants on the chamber surface. No significant treatment effects were observed when pulmonary flow resistance, tidal volume, respiratory frequency, and oxygen consumption were measured in guinea pigs, mice, or rats at 16-week intervals during the chronic exposures to exhaust. Exhaust-exposed mice showed a decrease in running activity for the first few weeks of exposure but then recovered to attain control levels. Decreases in mouse fertility rate and infant survival rate occurred in the exhaust chambers. This effect was confirmed in a second experiment.[119] Also, there was an increase in the rate of spontaneous pulmonary infection in exhaust-exposed animals. There were no significant effects of exhaust exposure on mortality, histopathology, growth rate, or hematologic indices.

Wayne and Chambers[205] reviewed studies on experimental animals exposed throughout their lifetimes to ambient Los Angeles atmosphere. Control animals were kept in rooms that were ventilated with special filters that removed most of the ambient air pollutants. The following maximal peak concentrations were recorded (ranges of the four exposure stations are given): 29 to 72 ppm CO, 50 to 121 pphm NO, 49 to 73 pphm NO_2, and 46 to 82 pphm oxidant. No clear evidence of chronic injury from the ambient air pollution was observed. However, there was suggestive evidence from pulmonary function tests, electron-microscopic examinations, and the incidence of pulmonary adenomas that aged animals had been adversely affected by ambient smog and that some reversible changes in pulmonary function of guinea pigs were noted during periods of peak air pollution. Increased 17-ketosteroid excretion suggested that breathing polluted ambient air was stressful for guinea pigs. The reported effects were marginal, and some of them may have been due to variations in temperature and humidity.

Beagles were exposed[120,200] to various pollutant mixes for 16 hr/day, 7 days/week for 61 months, and measurements of cardiovascular parameters and pulmonary function were made periodically.

TABLE 8-4. PULMONARY EFFECTS OF OZONE: PULMONARY FUNCTION

Ozone, $\mu g/m^3$	Ozone, ppm	Length of exposure	Observed effect(s)	Species	References
1960	1	3 hr	1-3 days post exposure, reduced vital capacity; 7 days post exposure, vital capacity reduced only slightly. A decrease in lung elasticity back to near normal conditions 3 days post exposure.	Rabbits	Yokoyama[212,213]
1960	1	6 hr/day 3-4 days	Increased residual volume/total lung capacity, functional residual capacity/total lung capacity, and total lung resistance; decreased chest wall resistance. No change in flow volume curves.	Rabbits	Yokoyama[214]
882	0.45	6 hr/day 6 days/week 6-7 weeks	No change in pulmonary pressure-volume relationship or ratio of collagen to elastin	Rats	Yokoyama and Ichikawa[216]
510 980 1960	0.26 0.5 1.0	2 hr 6.5 hr	Increased pulmonary flow resistance with increasing O_3 levels. Vital capacity not affected. Reduced diffusion capacity shown by some cats.	Cats	Watanabe et al.[204]
980	0.5	2 hr	Increased air current resistance and frequency of respiration, decreased tidal volume.	Guinea pigs	Yokoyama[211]
392	0.2	Continuous 30 days	Increase in lung volume and alveolar dimensions and a reduction in lung elasticity.	Young rats	Bartlett et al.[9]
666-2646	0.34-1.35	2 hr	Increased respiratory frequency and decreased tidal volume.	Guinea pigs	Murphy et al.[146]

TABLE 8-5. PULMONARY EFFECTS OF OZONE: EDEMA AND TOLERANCE

Ozone, $\mu g/m^3$ pre-exposure	Ozone, ppm pre-exposure	Length of pre-exposure	Ozone, $\mu g/m^3$ after latent period	Ozone, ppm after latent period	Length of exposure after latent period	Observed effect(s)	Species	Reference
490	0.25	6 hr	1966	1	6 hr	No tolerance to edema unless pretreated with methylprednisolone.	Rats	Alpert et al.[5]
588	0.3	1 hr	39,200	20	2 hr	Tolerance not developed by thymectomized animals, but developed by sham-operated animals.	Mice	Gregory et al.[93]
588	0.3	3 hr	588	0.3	3 hr	20% lower mortality for pre-exposed mice than mice receiving only one O_3 dose. Partial tolerance probably due to inhibition of edemagenesis.	Mice	Coffin and Gardner[37]
588-980	0.3-0.5	4 days	980, 1372, 1960	0.5,0.7, 1.0	1,2,4 days	Lack of total protection indicated by increased numbers of Type 2 cells.	Rats	Evans et al.[57]
980	0.5	6 hr				Edema as measured by recovery of ^{132}I in pulmonary lavage fluid.	Rats	Alpert et al.[5]
980	0.5	3 hr	43,120	22	3 hr	Using unilateral lung exposure technique, tolerance to edema a local effect and seen only in the pre-exposed lung.	Rabbits	Alpert et al.[4]
980	0.5	3 hr	5880 or 43,120	3 and 22	3 hr	Using unilateral lung exposure technique, tolerance developed only to pulmonary edema. No tolerance to the chemotaxis of polymorphonuclear leukocytes or decreased lysosomal hydrolase enzyme activity.	Rabbits	Gardner et al.[79]
1470	0.75	3 days	7840	4.0	8 hr	A smaller decrease in activities of glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase and levels of reduced glutathione in lungs of tolerant animals as compound to nontolerant animals.	Rats	Chow[27]
1960	1	1 hr				All animals X-irradiated to 800 R. 60% of O_3-pre-exposed mice survived. 100% of controls died.	Mice	Hattori et al.[99]
1960	1	1 hr	3920	2	1 hr	Tolerance to allergic response to inhaled acetylcholine.	Guinea pigs	Matsumura et al.[124]

Treatment groups are shown in Table 8-10. There were no specific abnormalities in cardiovascular function attributable to air pollution exposure during the exposure period. Pulmonary function measurements of the beagles were made after 18,[200] 36,[120] and 61[120] months of exposure. After 18 months, there were no differences in CO diffusing capacity (DL_{CO}), dynamic lung compliance (C_{Ldyn}), or total expiratory pulmonary resistance.

After 36 months of exposure, no statistically significant effects were observed, although there was an increased frequency of abnormal measurements in those animals (Group 7) receiving nitrogen oxides. By 61 months, more changes were evident, but only those related to oxidant exposure will be described here. Dogs exposed to irradiated auto exhaust (Group 3) had a higher ($p < 0.05$) mean single breath nitrogen washout than control animals. When the data were analyzed differently based on the number of animals showing alterations, it was found that more animals of groups 3 and 6 had higher ($p < 0.0001$) total expiratory resistances than the comparable control animals in groups 1 and 4.

Pulmonary biochemical measurements, made by Orthoefer et al.[158] on these dogs after a 2½-year

TABLE 8-6. EXTRAPULMONARY EFFECTS OF OZONE: HEMATOLOGY AND SERUM CHEMISTRY

Ozone, μg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
1960	1	30 min	Increased resistance to erythrocyte hemolysis.	Mice	Mizoguchi et al.[138]
1960	1	30 min	Increased resistance to erythrocyte hemolysis.	Mice	Christensen and Giese[33]
1686	0.86	8 hr/day 5 days/week 6 months	Increased infestation and mortality after infection with *P. berghei*. Increased acid resistance of erythrocytes.	Mice	Schlipkoter and Bruch[182]
1568	0.8	Continuous 29 days	Increased lysozyme activity by day 3.	Rats	Chow et al.[28]
980	0.5	Continuous 23 days	Increased hemolysis of erythrocytes of animals depleted of vitamin E. No such change when rats received vitamin E supplements.	Rats	Menzel et al.[132]
980	0.5	8 hr/day 7 days	No change in level of GSH or activities of GSH peroxidase, GSH reductase, or G-6-PD in erythrocytes.	Monkeys, rats	Chow et al.[30]
784	0.4	6 hr/day 5 days/week 10 months	Decreased serum albumin concentration; increased concentration of α and γ globulins; not much change in β globulin; no change in total serum proteins.	Rabbits	P'an and Jegier[164]
784	0.4	6 hr/day 5 days/week 6 months	No change in serum trypsin inhibitor capacity.	Rabbits	P'an and Jegier[165]
784	0.4	6 hr/day 5 days/week 10 months	Increase in serum protein esterase.	Rabbits	Jegier[110]
784	0.4	10 months	Increase in serum protein esterase.	Rabbits	P'an and Jegier[166]
392	0.2	8 hr/day 5 days/week 3 weeks	Increase in serum glutamic pyruvic transaminase, and hepatic ascorbic acid. No change in blood catalase.	Murine	Veninga[201]
392	0.2	60 min	Small decrease in total blood serotinin.	Rabbits	Veninga[202]
392	0.2	2 hr	Binucleated lymphocytes in blood doubled in number after exposure.	Murine	Veninga[202]
392	0.2	5 hr	Circulating blood lymphocytes had chromosome breaks up to 2 weeks post O_3. Additive effect with radiation exposure.	Hamsters	Zelac et al.[217,218]
294	0.15	5 hr	No increase in chromosomal aberrations.	Mice, hamsters	Gooch et al.[92]
451	0.23	5 hr			
1960	1.00	2 hr			
110	0.056	93 days	Decrease in whole blood cholinesterase, which returned to normal 12 days after exposure ceased.	Rats	Eglite[53]

TABLE 8-7. EXTRAPULMONARY EFFECTS OF OZONE: CENTRAL NERVOUS SYSTEM AND BEHAVIOR

Ozone, µg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
1960	1	Continuous 7 days	Reduction in voluntary activity.	Rats	Fletcher et al.[68]
1176	0.6	30 min	Avoidance response to O_3.	Mice	Peterson et al.[167]
980-1960	0.5-1	1 hr	Depression of evoked response to flash.	Rats	Xintaras et al.[210]
980	0.5	30 min	Elevation of simple and choice reactive time.	Subhuman primates	Reynolds and Chaffee[172]
118	0.06	—	Depression of gross motor activity as O_3 concentration increased.	Rats	Konigsberg and Bachman[113]
110	0.056	Continuous 93 days	No behavior change. No change in chronaxial ratios in muscles.	Rats	Eglite[53]

TABLE 8-8. EXTRAPULMONARY EFFECTS OF OZONE: MORPHOLOGY

Ozone, µg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
1470	0.75	4-8 hr	Morphological alterations of parathyroid gland.These changes reverted to normal 66 hr after exposure.	Rabbits	Atwal and Wilson[7]
392	0.2	5 hr/day 3 weeks	Structural changes in cell membranes and nuclei of myocardial muscle fibers that were reversible about 1 month following exposure.	Mice	Brinkman et al.[20]

TABLE 8-9. EXTRAPULMONARY EFFECTS OF OZONE: MISCELLANEOUS

Ozone, µg/m³	Ozone, ppm	Length of exposure	Observed effect(s)	Species	Reference
1960	1	3 hr/day up to 7 days	After 2 or 3 days of exposure, mice slept longer after injections of sodium pentobarbital.	Mice	Gardner et al.[78]
1960	1	90 min	No change in liver cytochrome P_{450} level.	Rabbits	Goldstein et al.[88]
1960	1	7- to 24-hr/day 18 months	In brain tissue, decreased COMT and altered MAO activity; no lipid peroxidation and no change in catecholamine levels, 5-nucleotidase, acetylcholinesterase, or ATPase.	Dogs	Trams et al.[199]
1568	0.8	5 days/week 18 weeks	Lower titratable acidity in urine on days 91, 98 and 112, and a higher pH on days 98 and 112. No change in urinary creatinine, creatine, uric acid/creatinine or amino acid nitrogen/creatinine.	Rats	Hathaway and Terrill[98]
490	0.25	2 hr	No differences in gastric secretomotor activity.	Rats	Roth and Tansy[178]
392	0.2	7 hr/day 5 days/week during gestation and 3 weeks after birth	Increased incidence of blepharophimosis and unlimited incisor growth.	Mice	Veninga[201]
110	0.056	Continuous 93 days	Increased levels of urinary 17-ketosteroids that remained elevated after exposure. Decreased ascorbic acid content of adrenal gland, which returned to normal levels 15 days after exposure ceased.	Rats	Eglite[53]

recovery period, showed no significant changes in collagen:protein ratios. However, prolyl hydroxylase (thought to be the rate-limiting enzyme in collagen synthesis) levels were elevated ($p < 0.05$) most in the lungs of animals exposed to irradiated exhaust (Group 3).[158]

Emik et al.[56] exposed various species of laboratory animals (mice, rats, and rabbits) to ambient California air for 2.5 years. The average ambient concentrations were 0.057 ppm oxidant, 1.7 ppm CO, 2.4 ppm hydrocarbons (as carbon), 0.019 ppm NO_2, 0.015 ppm NO, and 4.2 ppb PAN. The following results were obtained: Reduced pulmonary alkaline phosphatase in rats, reduced serum glutamic oxaloacetic transaminase in rabbits, increased pneumonitis in mice, increased mortality in male mice but not in female mice, reduced body weights in mice, and decreased running activity of male mice. When aging guinea pigs (24 months old) that lived in smog for 2 years were allowed to recover for 6 weeks in clean, filtered air and then exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone for 10 min, they had a smaller increase in lung resistance following ozone exposure than animals that lived in clean air before the same ozone exposure. The authors suggest that a possible adaptation occurred. Pulmonary tumors were found in the lungs of some mice, but there was no significant induction of lung adenomas in the two strains of mice exposed to ambient, smog-containing air. They state further that the exposure was probably near the threshold of effect, and therefore measurement of differences induced by smog was difficult.

The spontaneous activity of mice was reduced when the mice were exposed to smog ozone + gasoline vapor for 24 hr. Decreases ($p < 0.05$) were observed at 980 $\mu g/m^3$ (0.05 ppm) ozone. A simultaneous measurement of 1.69 ppm oxidant was made. As the concentrations of ozone and oxidant were increased (3650 $\mu g/m^3$, or 1.86 ppm ozone and 6.26 ppm oxidant), greater decreases in spontaneous activity were observed.[18] Emik and Plata[55] made a similar study of mice exposed continuously either to filtered air or to ambient air (oxidant from 0.062 to 0.239 ppm; NO_2 from 0.03 to 0.07 ppm; total hydrocarbons from 2.7 to 4.4 ppm) for 13 months. Animals exposed to the ambient air had decreased running activity compared to controls over the course of the study. The differences in activity were strongly related to oxidant for weekly intervals. High temperature and age were related to decreased activity in both groups.

Kotin and Thomas[116] exposed mice of both sexes continuously for 19 weeks to smog (formed by reacting gasoline with ozone, average of 1.25 ppm oxidant); natural urban atmosphere (fluctuated, highest reading recorded was 0.4 ppm oxidant); or clean, filtered air. Females held in the smog chamber had a decrease in conception rate ($p = 0.02$). The significant difference in litter rate in the smog-exposed animals was primarily attributed to an effect on the females. The survival rate of the newborns was also decreased ($p < 0.01$) when the parents were exposed to smog. Those animals in the smog chamber also had lower ($p < 0.05$) average litter sizes.

TABLE 8-10. ATMOSPHERIC MEAN CONCENTRATIONS AND THEIR STANDARD DEVIATIONS ADMINISTERED FROM 8 a.m. TO MIDNIGHT EACH DAY

		Pollutant, mg/m^3						
Group	Atmosphere	CO	HC (as CH_4)	NO_2	NO	O_x (as O_3)	SO_2	H_2SO_4
1	Control air	—	—	—	—	—	—	—
2	Nonirradiated auto exhaust	112.1±11.5	18.0±2.9	0.09±0.04	1.78±0.52	—	—	—
3	Irradiated auto exhaust	108.6±22.5	15.6±4.0	1.77±0.68	0.23±0.36	0.39±0.18	—	—
4	SO_2 + H_2SO_4	—	—	—	—	—	1.10±0.57	0.09±0.04
5	Nonirradiated auto exhaust + SO_2 + H_2SO_4	113.1±15.9	17.9±2.8	0.09±0.06	1.86±50.54	—	1.27±0.61	0.09±0.04
6	Irradiated auto exhaust + SO_2 + H_2SO_4	109.0±22.8	15.6±3.9	1.68±0.68	0.23±0.36	0.39±0.16	1.10±0.56	0.11±0.04
7	Nitrogen oxides	—	—	1.21±0.22	0.31±0.08	—	—	—
8	Nitrogen oxides	—	—	0.27±0.62	2.05±0.26	—	—	—

Nakajima et al.[152] studied histopathologic changes in the lungs of mice that were exposed to irradiated auto exhaust and oxidant-fortified exhaust-gas mixtures for 2 to 3 hr/day, 5 days/week for a month. Histopathologic changes resembling tracheitis and bronchial pneumonia were observed in mice exposed to atmospheres containing oxidant at 0.1 to 0.5 ppm. In those exposed to atmospheres containing 0.1 to 0.15 ppm, the changes were minimal, the main finding being irregular arrangement of the epithelial cells of the relatively thick bronchioles.

Summary

Animal toxicological studies have investigated the biological response of laboratory animals to sample atmospheres of photochemical reaction mixtures. Long-term exposure of various species of animals to ambient California atmospheres have produced changes in pulmonary functional measurements in the guinea pig (980 $\mu g/m^3$, or 0.5 ppm) and a number of biochemical and pathological effects in mice, rats, and rabbits.

Exposure to irradiated auto exhaust containing oxidant levels ranging from 0.2 to 1.0 ppm also produced numerous changes in experimental animals. In mice, an increase in susceptibility to infection and a decrease in spontaneous running activity, infant survival rate, and fertility were reported in exhaust-exposed animals. In guinea pigs, the following changes were observed: Increased tidal volume, increased minute volume, increased flow resistance, and a decreased frequency of breathing after short-term exposure to irradiated auto exhaust. Dogs exhibited several alterations in normal pulmonary function capabilities.

Note that experimental exposure to irradiated auto exhaust usually involves variable concentrations of carbon monoxide, hydrocarbons, and nitrogen oxides, as well as oxidants. The studies of the effects of oxidants on animals are summarized in tabular form in Table 8-11.

TABLE 8-11. EFFECTS OF OXIDANTS ON ANIMALS

Pollutant concentration	Length of exposure	Observed effect(s)	Species	Reference
O_3 (980 $\mu g/m^3$; 0.5 ppm) and gasoline vapor (oxidant 1.69 ppm)	24 hr	Decreased spontaneous running activity	Mice	Boche and Quilligan[18]
O_3 and gasoline (oxidant 1.25 ppm)	Continuous 19 weeks	Decreased conception rate, litter rate, and newborn survival.	Mice	Kotin and Thomas[116]
Irradiated auto exhaust (oxidant, 0.1-0.5 ppm)	2-3 hr/day 5 days/week for 1 month	Tracheitis and bronchial pneumonia.	Mice	Nakajima et al.[152]
Oxidant, 0.062-0.239 ppm; NO_2, 0.03-0.07 ppm; hydrocarbons, 2.7-4.4 ppm	Continuous 13 months	Decreased spontaneous running activity.	Mice	Emik and Plata[55]
Oxidant, 0.057 ppm; CO, 1.7 ppm; hydrocarbons, 2.4 ppm; NO_2, 0.019 ppm; NO, 0.015 ppm; PAN, 4.2 ppb	Continuous 2.5 years	Reduced pulmonary alkaline phosphatase (rats); reduced serum glutamic oxaloacetic transaminase (rabbits); increased pneumonitis (mice); increased mortality (male mice); reduced body weights (mice); decreased running activity (male mice); no significant induction of lung adenomas (mice).	Mice, rats, rabbits	Emik et al.[56]
7 exposure groups (see Table 8-10); various mixtures of CO, hydrocarbons, NO_2, NO, O_3, SO_2, H_2SO_4, and irradiated and nonirradiated auto exhaust	16 hr/day, 7 days/weeks for 68 months	No changes in cardiovascular parameters attributable to pollutants; alterations in pulmonary function.	Beagles	Vaughan et al.[200] Lewis et al.[120]

EFFECTS OF PEROXYACETYLNITRATE ON EXPERIMENTAL ANIMALS

Experimental Data

To determine the effects of PAN on acute respiratory infections, Thomas et al.[197] exposed mice to PAN for 2 or 3 hr and then to aerosols of *Streptococcus pyogenes* (Group C). The data indicate that a 3-hr exposure to 26,000 $\mu g/m^3$ (5.2 ppm) PAN resulted in an increased ($p < 0.05$) incidence of fatal pulmonary infection.

Campbell et al.[24] exposed mice to PAN, 485,000 to 725,000 $\mu g/m^3$ (97 to 145 ppm) as measured at the chamber outlet, for 2 hr at 80°F (27°C). The studies demonstrated that most mice exposed to 550,000 $\mu g/m^3$ (110 ppm) or more PAN died within a month, and mice exposed to higher concentrations died earlier. Median lethal exposures characteristically produced a delayed mortality pattern, with most deaths occurring during the second and third weeks after exposure. Mortality was greater among older than among younger mice. It was greater at higher temperatures, but was not influenced appreciably by changes in relative humidity.

Dungworth et al.[51] exposed mice to 75,000 $\mu g/m^3$ (15 ppm) PAN for 6 hr/day, 5 days/week for 26 weeks. Exposed animals showed a 28-percent reduction in body weight ($p < 0.001$) and a 26-percent increase in lung-heart weight ($p < 0.001$). Histological examination revealed chronic hyperplastic bronchitis and proliferative peribronchiolitis. Bronchi developed various degrees of epithelial hyperplasia, and sometimes metaplasia. Approximately 50 percent of the lungs had foci of squamous metaplasia in the trachea or major bronchi. The most noteworthy feature of the hyperplastic bronchial response was the tendency for acinar structures to form within the bronchial walls. In the bronchial epithelium, cilia were frequently absent, and there was a severe reduction in mucus-secreting cells. There was no evidence of the accumulation of perivascular and peribronchiolar lymphocytes and plasma cells. The most conspicuous bronchiolar alteration was bronchiolectasis. Peribronchiolar epithelial hyperplasia or metaplasia was often accompanied by cellular accumulations, principally of monocytes. Some alveoli had increased numbers of Type 2 cells and nuclei (probably of Type 1 cells). There was also some dilatation of alveolar ducts and breakdown of walls in peribronchiolar regions, usually in association with epithelial hyperplasia.

Summary

Short-term exposures to PAN in excess of 26,000 $\mu g/m^3$ (5.2 ppm) have been found to result in increased mortality of mice challenged with aerosols of viable bacteria. Longer-term exposures to 75,000 $\mu g/m^3$ (15 ppm) resulted in pulmonary morphological alterations.

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9. CLINICAL APPRAISAL OF THE EFFECTS OF OXIDANTS

OCCUPATIONAL AND ACCIDENTAL EXPOSURES TO OZONE

Schonbein,[55] the discoverer of ozone, was probably the first person occupationally exposed to this gas. His description in 1851 of subjective symptoms and basic characteristics of ozone was very complete. Dadlez[11] in 1928 found that 2940 μg/m³ (1.5 ppm) ozone rendered the atmosphere intolerable. He cites an investigation by D'Arsonval who found that 780 μg/m³ (0.4 ppm) ozone in the atmosphere produced symptoms of discomfort and irritation within about 30 min. after exposure began. In 1931, Flury and Zernik[14] described the following symptoms as being characteristic of increasing exposure to ozone: 920 μg/m³ (0.47 ppm) causes distinct irritation of mucous membranes, and 1840 μg/m³ (0.94 ppm) causes sleepiness in 1 hr; at higher concentrations, ozone causes increased pulse rate, sleepiness, and prolonged headache. Ozone exposure may also result in dyspnea (difficult breathing) and pulmonary edema.

Kleinfeld et al.[44] and Kleinfeld[43] reported several cases of severe ozone intoxication in welders using a consumable electrode technique, which was new at that time (1957). Three plants were investigated, and in all cases, the ozone concentration was monitored at the breathing zone of the consumable electrode machine. In the first plant, an ozone concentration of 490 μg/m³ (0.25 ppm) was found. The workers had no complaints, and clinical findings were noncontributory. In the second plant, the ozone concentration ranged from 590 to 1570 μg/m³ (0.3 to 0.8 ppm). Two of the four welders complained of chest constriction and throat irritation. Clinical examination disclosed no abnormalities. In the third welding plant, the ozone concentration was 17,990 μg/m³ (9.2 ppm). Also, a trichloroethylene degreaser was located about 50 ft from the welding area. Concentrations of nickel carbonyl and nitrogen oxides were found to be negligible, and tests for phosgene in the welding area were negative. The authors gave case histories of three workers (although there were others) who also complained of severe headaches, throat irritation, and lassitude. One of the cited patients developed severe dyspnea and substernal oppression and, on admission to a hospital, was found to be in pulmonary edema. Chest X-rays showed diffuse peribronchial infiltration consistent with peribronchial pneumonia. The patient made a slow recovery over a 2-week period but still complained of fatigue and exertional dyspnea after 9 months. The other two workers had similar but less severe symptoms.

Challen et al.[10] performed a similar clinical and environmental survey. Ozone concentrations of 1570 to 3330 μg/m³ (0.8 to 1.7 ppm) were found. Eleven of 14 workers who were directly involved in welding complained of respiratory symptoms. No further symptoms were reported when ozone concentrations were reduced to 390 μg/m³ (0.2 ppm). The results of this study, however, were complicated by the fact that concentrations of trichloroethylene up to 1275 μg/m³ (0.638 ppm) were also present.

In 1965, Kelly and Gill[38] reported a case of severe, accidental ozone poisoning in which the worker could have been exposed to concentrations as high as 21,950 μg/m³ (11.2 ppm) for up to 120 min. The symptoms he developed were so severe that he almost lost consciousness. He perspired, coughed continuously, and had a decrease in blood pressure that was accompanied by a weak and accelerated pulse. Administration of oxygen relieved his symptoms rather quickly, and 48 hr after the incident, he was completely asymptomatic.

Young et al.[61] made the first study of pulmonary function changes in workers exposed to ozone. They studied seven men, all smokers, who were engaged in argon-shielded electric arc welding. The concentration of ozone in the welding shop was estimated by the rubber cracking technique and found to be 390 to 590 μg/m³ (0.2 to 0.3 ppm).

The following measurements of pulmonary function were made: Vital capacity (VC), functional residual capacity (FRC), maximal midexpiratory flow rate (FEF 25 to 75), 0.75-second forced expiratory volume ($FEV_{0.75}$), and carbon monoxide diffusing capacity (DL_{CO}) at rest and at exercise. No convincing evidence was found that functional impairment developed in association with long-term exposure to 390 to 590 $\mu g/m^3$ (0.2 to 0.3 ppm) ozone in these seven smokers.

The available data on occupational exposures of humans to ozone are summarized in Table 9-1.[10,11,14,38,43,44,61]

TABLE 9-1. SUMMARY OF AVAILABLE DATA ON OCCUPATIONAL EXPOSURE OF HUMANS TO OZONE[10,11,14,38,43,44,61]

Ozone, $\mu g/m^3$	Ozone, ppm	Subjective complaints	Clinical findings attributed to ozone	Measurements of pulmonary function	Other comments	Reference
490	0.25	None	None	None		Kleinfeld et al.[44]
590-1570	0.3-0.8	Chest constriction and throat irritation in 2 to 4 subjects.	None	None		Kleinfeld et al.[44]
17,990 (peak concentration)	9.2 (peak concentration)	Severe headaches, throat irritation, and lassitude in 7 or 8 subjects.		None	Negligible nickel carbonyl and oxides of nitrogen. Trichloroethylene degreaser located 50 ft from welding area. Tests for phosgene, negative.	Kleinfeld et al.[44] Kleinfeld[43]
		Cough, choking, dyspnea, and substernal oppression in 3 of 8 subjects.	By X-ray, molted densities in both lungs, clearing after 9 days.	None		
		Very severe headache: dyspnea. Substernal oppression in 1 of 8 subjects.	Severe pulmonary edema. By X-ray peribronchial infiltration consistent with peribronchial pneumonia.	None		
390	0.2		None	None		Challen et al.[10]
1570-3330	0.8-1.7	Dry mouth and throat, irritation of nose and eyes, detection of disagreeable smell in 11 of 14 subjects.	None	None	Concentration of trichloroethylene up to 238 ppm found.	Challen et al.[10]
390-590	0.2-0.3	Detection of irritating odor; soreness of eyes and dryness of mouth, throat, and trachea in 1 of 7 subjects.	None	VC decreased in 3 of 7 subjects. FRC decreased in 2 of 7 subjects. DL_{CO} decreased in 1 of 7 subjects.	All decreases in pulmonary function measurements were small. All subjects were smokers.	Young et al.[61]
780	0.4	Discomfort and irritation in about 30 min.	None	None		Dadlez[11] (D'Arsonval)
920	0.47	Distinct irritation of mucous membranes.	None	None		Flury and Zernik[14]
1,840	0.94	Coughing, irritation, and exhaustion, within 1½ hr.	None	None		Flury and Zernik[14]
5,900	3.0	Sleepiness within 1 hr.	None	None		Flury and Zernik[14]
21,950	11.2	Profuse perspiration; continual coughing; decreased blood pressure; weak, accelerated pulse.	None	None	Severe symptoms almost caused subject to lose consciousness.	Kelly and Gill[38]

CONTROLLED STUDIES OF HUMAN HEALTH EFFECTS

Studies conducted before 1970 may be summarized as follows.

Under experimental conditions, repeated exposure of humans to ozone produced the following effects:

1. No apparent effects on pulmonary function were observed at concentrations up to 390 μg/m³ (0.2 ppm) for 3 hr/day, 6 days/week for 12 weeks.[6]
2. The threshold level at which nasal and throat irritation will occur appears to be about 590 μg/m³ (0.3 ppm).[61]
3. Concentrations of 980 μg/m³ (0.5 ppm) have caused a 20-percent decrease in the 1-sec forced expiratory volume (FEV_1) observed after 8 weeks of intermittent exposure (3 hr/day, 6 days/week); this change returned to normal during the post-exposure period of 6 weeks.[6]

In Table 9-2,[6,20,25,31,36,60] the results of experimental appraisal of short-term exposures to ozone are shown as follows:

1. Some subjects have shown small but statistically significant increases in airway resistance after 1 hr of (presumably resting) exposure to 200 μg/m³ (0.10 ppm) ozone.[20] However, the significance of this finding is unclear, since increases in resistance were small, and since the same subjects showed smaller, nonsignificant increases in resistance after exposure to 780 μg/m³ (0.4 ppm) ozone than they had shown after exposure to 200 μg/m³ (0.1 ppm).
2. Concentrations of 200 to 780 μg/m³ (0.1 to 0.4 ppm) for 1 hr have been shown to increase airway resistance (Raw) slightly, but adequate information for this concentration range is lacking.[20]
3. Exposure to a concentration of 1960 μg/m³ (1.0 ppm) for periods of 1 to 2 hr produced changes in pulmonary function. These were increased airway resistance, decreased vital capacity, decreased carbon monoxide diffusing capacity, and decreased forced expiratory volume.[20]
4. One individual was unable to tolerate concentrations of 1960 to 5880 μg/m³ (1.0 to 3.0 ppm) over a period of about 2 hr. Extreme fatigue and lack of coordination were experienced.[25]
5. Concentrations of about 17,640 μg/m³ (9.0 ppm) produced severe pulmonary edema and possible acute bronchiolitis.[36]

Details of selected controlled human studies reported after 1970 are discussed below.

Bates et al.[3,4] measured significant changes in lung function: A decrease (a) in maximal flow rate at 50 percent of the vital capacity (FEF 50 percent) and (b) in maximal transpulmonary pressure (PstTLC); and an increase in total pulmonary resistance (RL) in 10 normal male subjects aged 23 to 53 years (including two smokers) exposed to pure ozone at 1470 μg/m³ (0.75 ppm) for 2 hr. Two of the three subjects who exercised intermittently at twice the resting volume showed accentuated effects. In a separate study by Hazucha et al.[35] on the effects of short-term exposure, significant decreases in forced vital capacity (FVC), maximal midexpiratory flow rates, FEF 25 to 75 percent, FEV_1, and FEF 50 percent, and increases in closing capacity (CC) and residual volume (RV) were found in 12 normal young males (including six smokers) exposed to pure ozone at 1470 and 730 μg/m³ (0.75 and 0.37 ppm) for 2 hr during alternating rest and exercise periods. The higher concentration affected smokers more than nonsmokers, whereas at the lower concentration, the reverse was found. In these two studies, most subjects complained of cough, chest tightness, and substernal soreness. A few also had pharyngitis, dyspnea, and wheezing. In another study,[2,32] all mean dynamic lung function parameters in 10 nonsmokers and 10 smokers showed a progressive decrease with continuation of exposure, returning close to pre-exposure levels after 2 hr of recovery (Figure 9-l).[32] Average minute ventilation changed only slightly during the exposure, although the frequency of breathing increased, and the tidal volume decreased steadily with the duration of exposure (Figure 9-2).

Bates and Hazucha[2] and Hazucha and Bates[33] reported a marked decrease in pulmonary function among healthy subjects performing light exercise while exposed to 730 μg/m³ (0.37 ppm) of ozone and 1000 μg/m³ (0.37 ppm) of sulfur dioxide for 2 hr. Throat irritation, coughing, and chest pain were also experienced. These effects, as well as the decrease in lung function, occurred after exposure to the mixture for periods as short as one-half to 1 hr and persisted for several hours after termination of the exposure. Ozone concentrations were measured by a Mast meter with CrO_3 scrubber for SO_2 removal, and SO_2 levels were monitored with a conductimetric analyzer. To investigate further the possible interactive effects of $SO_2 + O_3$ and the mechanism of interaction, Bell et al.[5] initiated a series of controlled human-exposure studies. The studies of Bell et al. suggest less severe acute toxicity of mixtures of (730 $\mu g/m^3$) 0.37 ppm ozone and (1000 $\mu g/m^3$) 0.37 ppm SO_2 than Bates and Hazucha had previously observed. There are several possible explanations: (1) The Los Angeles residents had undergone biological adaptation to chronic pollutant exposure; (2) the Montreal subjects used in the experiment were not as reactive to the $SO_2 + O_3$ mixture when tested in Los Angeles as when tested in Montreal, possibly because of significantly higher concentrations of the sulfur compounds in the respirable aerosols that can form in the Montreal chamber. The background air in the Rancho Los Amigos studies was highly purified, whereas the sulfur aerosol concentration in the Montreal chamber during the mixed-gas exposures was similar to 2-hr sulfate aerosol concentrations during the worst pollution episodes in urban areas. The effects observed in the Montreal chamber probably more nearly parallel the health effects that might result during

TABLE 9-2. SUMMARY OF DATA ON HUMAN EXPERIMENTAL EXPOSURE TO OZONE BEFORE 1970[6,20,25,31,36,60]

Ozone, $\mu g/m^3$	Ozone, ppm	Length of exposure	No. and sex of subjects	Subjective complaints	Measurements of pulmonary function	Other comments	Reference
9,800-19,600	5-10	Not available	3 male	Drowsiness, headache.	None	Measurement of O_3 probably inaccurate.	Jordan and Carlson[36]
2,940-3,920	1.5-2	2 hr	1 male	CNS depression, lack of coordination, chest pain, cough for 2 days, tiredness for 2 weeks.	VC: Decreased 13%; returned to normal in 22 hr. FEV_{30}: Decreased 16.8% after 22 hr. MBC: Decreased very slightly.[a]		Griswold et al.[25]
390	0.2	3 hr/day 6 days/week for 12 weeks	6 male	None	VC: No change. $FEV_{1.0}$: No change.	0.66 upper respiratory infections/person in 12 weeks; Cf. control group had 0.95 in the same period. 0.80 upper respiratory infections/person in 12 weeks.	Bennet[6]
980	0.5	3 hr/day 6 days/week for 12 weeks	6 male	No irritating symptoms, but could detect ozone by smell.	VC: Slight but not significant decrease toward end of 12 weeks. Returned to normal within 6 weeks after exposure.	0.80 upper respiratory infections/person in 12 weeks.	
1,180-1,570	0.6-0.8	2 hr	10 male 1 female	Substernal soreness and tracheal irritation 6 to 12 hr after exposure, disappearing within 12 to 24 hr in 10/11 subjects.	DL_{CO}: Mean decrease of 25% (11/11 subjects). VC: Mean decrease of 10%, which was significant (10/10 subjects). $FEV_{0.75} \times 40$: Mean decrease of 10%, which was significant.		Young et al.[60]
					(10/10 subjects): FEF 25-75: Mean decrease of 15%, which was not significant. Mixing efficiency: No change (2/2 subjects). Airway resistance: Slight increase, but within normal limits. Dynamic compliance: No change (2/2 subjects).		Young et al.[60]

TABLE 9-2. (cont'd). SUMMARY OF DATA ON HUMAN EXPERIMENTAL EXPOSURE TO OZONE BEFORE 1970[6,20,25,31,36,60]

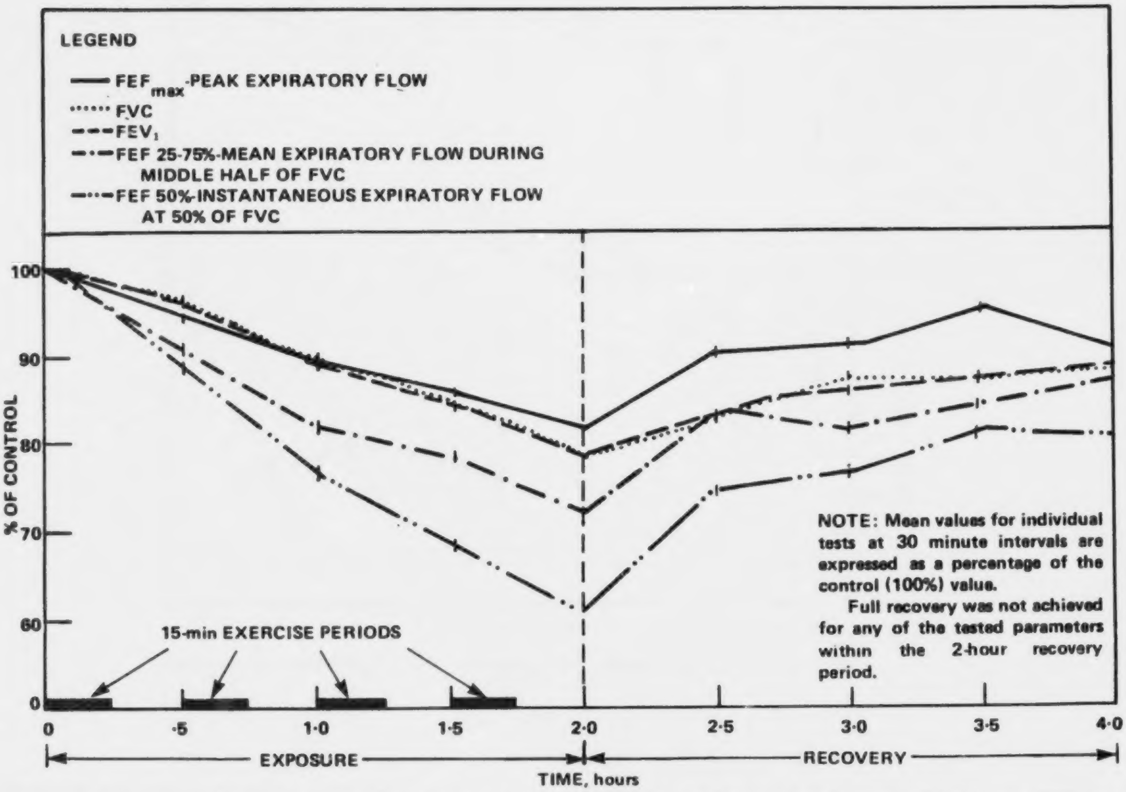
Ozone, $\mu g/m^3$	Ozone, ppm	Length of exposure	No. and sex of subjects	Subjective complaints	Measurements of pulmonary function	Other comments	Reference
Up to 7,800	Up to 4.0	10 to 30 min	11	Headache, shortness of breath, lasting more than 1 hr.	VC: Mean decrease of 16.5% (4/8 subjects showed decrease > 10%) $FEV_{1.0}$: Mean decrease of 20% (5/8 subjects showed decrease > 10%. FEF 25-75: Mean decrease of 10.5% (5/6 subjects showed a decrease). MBC: Mean decrease of 12% (5/8 subjects showed a decrease). DL_{CO}: Decrease of 20 to 50% in 7/11 subjects; increase of 10% to 50% in 4/11 subjects.	Only 5/11 subjects tolerated dose for full 30 min. Wide variations in DL_{CO}.	Hallett[31]
200	0.1	1 hr	4 male		Airway resistance: Mean increase of 3.3% at 0 hr after exposure (1/4 subjects showed an increase of 45%).	One subject had history of asthma and experienced hemoptysis 2 days after 1 ppm.	Goldsmith and Nadel[20]
780	0.4	1 hr	4 male	Odor	Airway resistance: Mean increase of 3.5% at 0 hr after exposure; (1/4 subjects showed an increase of 60%); mean increase of 12.5% 1 hr after exposure).		Goldsmith and Nadel[20]
1,180	0.6	1 hr	4 male	Odor	Airway resistance: Mean increase of 5.8% at 0 hr after exposure (1/4 subjects showed an increase of 75%); mean increase of 5% 1 hr after exposure.		
1,960	1.0	1 hr	4 male	Throat irritation and cough	Airway resistance: Mean increase of 19.3% at 0 hr after exposure (3/4 subjects showed an an increase of > 20%); mean increase of 5% 1 hr after exposure.		

[a] MBC = maximum breathing capacity

smog episodes in regions with high oxidant and sulfur pollution than do the effects observed in the Rancho Los Amigos chamber.

Studies by Hackney et al.[26,28,30] used basically the same rest or intermittent exercise protocol as that developed in the Montreal laboratory. To simulate summer exposure in the Los Angeles southern coastal basin, the additional stress of heat (31°C, or 88°F at 35 percent relative humidity) was included, and the same subjects were exposed several times to either ozone or mixtures of ozone with other pollutants. Careful attention was given to environmental control, pollutant monitoring and generation, and subject selection. Four male subjects, aged 36 to 49 years and judged by subjective criteria to have normally reactive airways (i.e., with no history of cough, chest discomfort, or wheezing), completed this protocol. As assessed by clinical response and measures of respiratory, cardiac, and metabolic functional change, no obvious effects were noted after exposure for 4 to 5 hr to 980 $\mu g/m^3$ (0.5 ppm) ozone, to a combination of 0.5 ppm ozone and 560 $\mu g/m^3$ (0.3 ppm) nitrogen dioxide, or to a mixture of 0.5 ppm ozone, 0.3 ppm nitrogen dioxide, and 35 $\mu g/m^3$ (30 ppm) carbon monoxide. A point of interest is that in a group of unreactive subjects, the same investigators observed substantial decrements in lung function after only 2 hr of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise. Another group of four, aged 29 to 41 years, who had previously experienced clinical bronchospasms and were judged by subjective criteria to have hyper-reactive airways, developed clinical discomfort and were unable to complete the protocol. Exposed to ozone at 980 $\mu g/m^3$ (0.5 ppm) for 4 to 5 hr, this group developed marked changes in pulmonary mechanics and gas distribution.

In later experiments, some effects were found after exposure of the same group of subjects to ozone at 730 $\mu g/m^3$ (0.37 ppm), but not after exposure at 490 $\mu g/m^3$ (0.25 ppm). A third group of seven subjects, aged 22 to 36 years and judged to have normally reactive airways, showed only minimal effects on the first day of exposure when exposed to ozone at 980 $\mu g/m^3$ (0.5 ppm) for 2 hr; however, five of the seven showed significant effects on a second exposure day. A fourth group of seven subjects, aged 22 to 41 years (three hyper-reactors and four normals) were exposed for 2 hr/day on 2 successive days in 3 consecutive weeks to 490 $\mu g/m^3$ (0.25 ppm) ozone, a mixture of 490 $\mu g/m^3$ (0.25 ppm) ozone and 560 $\mu g/m^3$ (0.3 ppm) nitrogen dioxide, or a mixture of 490 $\mu g/m^3$ (0.25 ppm) ozone, 560 $\mu g/m^3$ (0.3 ppm) nitrogen dioxide, and 35 mg/m^3 (30 ppm) carbon monoxide. These subjects showed no obvious effects. A fifth



NOTE: Mean values for individual tests at 30 minute intervals are expressed as a percentage of the control (100%) value.

Full recovery was not achieved for any of the tested parameters within the 2-hour recovery period.

Figure 9-1. Changes with time in dynamic lung function tests in nonsmokers during a 2-hr exposure to 0.75 ppm ozone and during recovery.[32]

group of five subjects, aged 27 to 41 years, was exposed to 730 $\mu g/m^3$ (0.37 ppm) ozone for 2 hr. No important changes with exposure were found in most physiologic measures. The experimental design in all these studies does not permit statistical analysis of the combined data; therefore, no firm conclusions can be drawn concerning dose-response relationships.

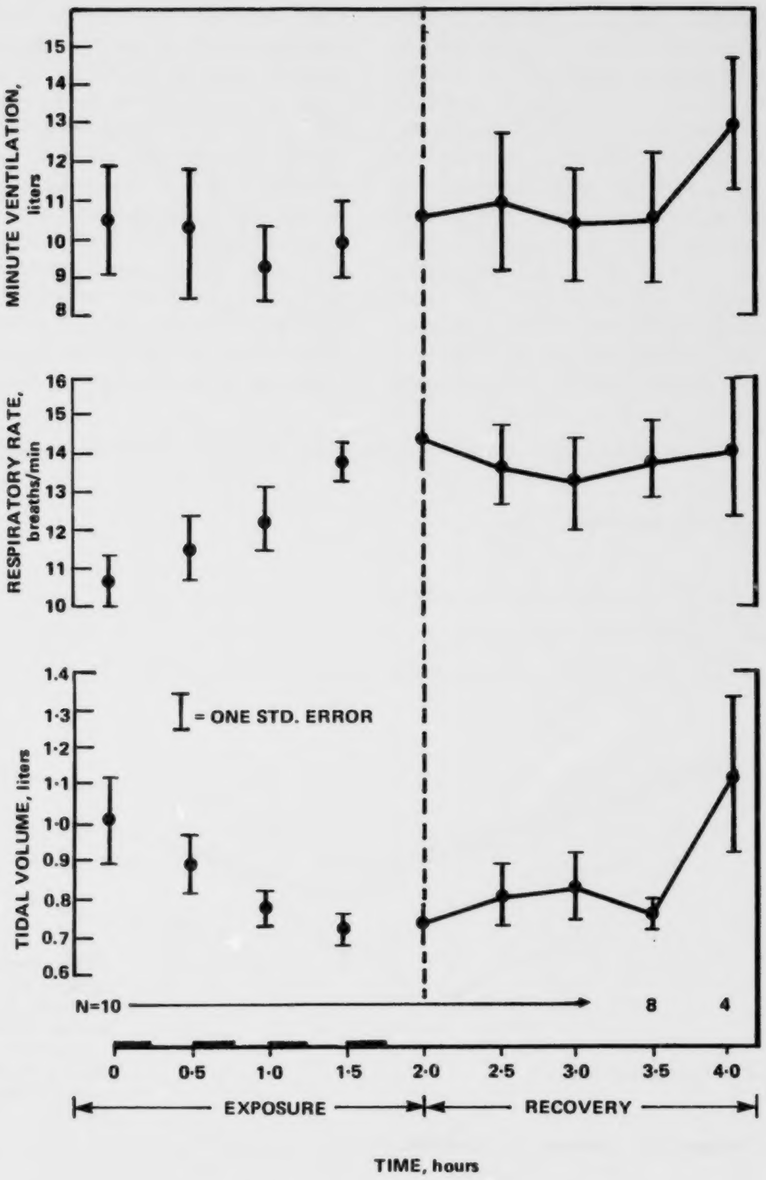


Figure 9-2. Changes with time of minute ventilation, respiratory rate, and tidal volume in ozone-exposure (0.75) and subsequent recovery period in exercising nonsmokers.[32] (Note: Heavy bars on the abscissa mark 15-min periods of light exercise.)

In studies by Kerr et al.,[39] 10 smokers and 10 nonsmokers, aged 21 to 60 years, were exposed to ozone at 980 $\mu g/m^3$ (0.5 ppm) for 6 hr in an environmental chamber. During this period, they engaged in two 15-min medium exercise sessions (100 watts) on a bicycle ergometer. Subjects who experienced typical symptoms were, in general, the ones who developed objective evidence of decreased pulmonary function. The most significant changes from control values for the group as a whole (20 subjects) after ozone exposure were observed in several pulmonary function tests—specific airway conductance (SGaw), pulmonary resistance (RL), and FVC. No significant change was observed with respect to diffusing capacity (DL_{CO}), static lung compliance (Cst), or the various tests derived from the single-breath nitrogen elimination rate. When the smokers were considered as a separate group, no significant decrease in pulmonary function was observed. The group of nonsmokers considered separately showed a significant decrease in dynamic compliance as well as in those parameters noted for all 20 subjects considered together. Prolonged exposure of four subjects to 0.5 ppm for 10 hr further decreased specific airway conductance.

Folinsbee et al.[17] tested the response of 28 subjects after ozone exposure to three stages of ergometer exercise with loads adjusted to 45, 60 and 75 percent of maximal aerobic power. The subjects were exposed to ozone at 730, 980, or 1470 $\mu g/m^3$ (0.37, 0.50, or 0.75 ppm) for 2 hr, at rest or while exercising intermittently. Subjects had 15 min of rest alternated with 15 min of exercise at a workload sufficient to increase ventilation by a factor of 2.5. At submaximal exercise, neither oxygen consumption nor minute ventilation was significantly altered after ozone exposure at any concentration. The primary response was an alteration of exercise ventilatory pattern. An increase in breathing rate ($r=0.98$) and a decrease in tidal volume ($r=0.91$) correlated well with the dose of ozone, calculated as the volume of ozone inspired during exposure. It was concluded that through its irritant properties, ozone modified normal ventilatory response to exercise and that this effect was dose-dependent.

In a second study, Folinsbee and associates[16] assessed the effect of exposure to 1470 $\mu g/m^3$ (0.75 ppm) ozone on the cardiac and pulmonary function of maximally exercising, healthy young adult males. During 2-hr exposures, subjects alternately rested for 15 min and lightly exercised for 15 min at a workload of about 50 watts. In random order, each subject also underwent a 2-hr control exposure to filtered air. At the conclusion of all exposures, subjects exercised maximally until exhausted. Values of the following parameters, measured during maximal exercise, were significantly lower after ozone exposure than after filtered air exposure: Maximum work load attained, heart rate, minute volume, tidal volume, and oxygen uptake. Respiratory frequency and the ratio of tidal volume to FVC during maximal exercise did not differ significantly following ozone versus filtered air exposures. The authors concluded that the decrease in maximal exercise performance after ozone exposure probably stemmed from respiratory rather than cardiac factors. In their minds, the most likely mechanism underlying the observed results is the stimulation by ozone of irritant receptors in the lung. Such stimulation in turn produces restriction of inspiratory reserve volume, reduction of tidal volume during maximum exercise and a modest increase in airway resistance.

A more specific study of airway irritability was conducted by Golden et al.[19] They showed that after a 2-hr exposure to 1180 $\mu g/m^3$ (0.6 ppm) ozone, bronchial reactivity (as assessed by response to histamine challenge) increased. Seven days following the exposure, bronchial sensitivity was still elevated, and in some subjects, hyperirritability persisted for up to 3 weeks. The authors believe that ozone damaged the airway epithelium and sensitized bronchial irritant receptors.

DeLucia and Adams[12] studied the effects of graded exercise on lung function and blood biochemistry in six men after 1 hr of exposure to 290 $\mu g/m^3$ (0.15 ppm) and 590 $\mu g/m^3$ (0.30 ppm) of ozone via a mouthpiece. The workloads were 25 percent, 45 percent, and 65 percent of each individual's maximum oxygen uptake ($\dot{V}O_2$ max). Ventilation volume and $\dot{V}O_2$ were unaffected by even the most severe exposure/exercise protocols. However, most subjects demonstrated signs of toxicity (symptoms such as congestion, wheezing, and headache) during the most stressful protocols. In addition, vital capacity, forced expiratory volume at 1 sec, and midmaximum flow rate decreased significantly after inhalation for 1 hr of 590 $\mu g/m^3$ (0.30 ppm) at 65 percent $\dot{V}O_2$ max. Discernible, though not statistically significant, changes in respiratory pattern were also observed after exposure to 290 $\mu g/m^3$ (0.15 ppm) ozone and exercise at 65 percent $\dot{V}O_2$ max. This study emphasizes the importance of actually quantifying the dose rather than merely defining the exposure level, as well as quantifying the differential effects that may result from mouth breathing compared to nose breathing.

The combined effects of different levels of exercise, heat stress, and ozone on lung function of 14 males were studied by Folinsbee et al.[15] Subjects were exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone for 2-hr periods under four environmental conditions: (1) 25°C (77°F), 45 percent RH; (2) 31°C (88°F), 35 percent RH; (3) 35°C (95°F), 40 percent RH; and (4) 40°C (104°F), 50 percent RH. One 30-min exercise period (40 percent of $\dot{V}O_2$ max) was included in every 2-hr exposure run. The results of this study show that besides well established physical factors such as concentration of ozone, duration of exposure, and intensity of exercise, the extent of lung functional response will depend also on timing of exercise into the exposure period, lapse time between end of exercise and testing period, relative humidity, and ambient air temperature.

Silverman et al.,[56] using the Montreal exposure protocol (intermittent exercise, 730 $\mu g/m^3$ [0.37 ppm] or 1470 $\mu g/m^3$ [0.75 ppm] ozone for 2 hr), basically confirmed the results obtained previously by Hackney et al.[26,28,30] and Bates and Hazucha.[2,19] Moreover, they showed a high linear correlation between the effective dose of ozone (calculated as concentration times the relative volume of ventilation) and percent changes in lung function measurements ($r = 0.70$ to 0.95). The reported data indicate that for a given effective dose, exposure to a high concentration for a short time is more effective than a longer exposure to a lower concentration.

Knelson et al.[45] exposed 22 male volunteers, aged 19 to 27, to ozone for up to 4 hr at 780 $\mu g/m^3$ (0.4 ppm). Subjects were sedentary during exposure except for two 15-min exercise periods on a bicycle ergometer at 700 kg-m/min exercise that about doubled the heart rate and quadrupled the minute ventilation. After 2 hr of ozone exposure, there was a significant change ($p < 0.05$) in FVC, midmaximal expiratory flow (MMEF), and airway resistance (Raw). Several other measures (FEV$_1$, $\dot{V}_{50}$, and $\dot{V}_{25}$) were lower after 2 hr of exposure, but the statistical significance was borderline. However, after 4 hr of exposure, all flow measures were significantly decreased, compared with controls. After 4 hr, Raw increased, FVC decreased further, and FEV$_1$ decreased significantly. Residual volume, functional residual capacity, and total lung capacity (TLC) did not change as a result of the ozone exposure. Ketcham et al.,[40] employing the same laboratory facilities as the previous investigators, also demonstrated significant deterioration of pulmonary function in 30 subjects. The average FEF 25 to 75 percent, which is the most sensitive test, decreased by almost 30 percent from the control value after 2 hr of exposure to 1180 $\mu g/m^3$ (0.6 ppm) ozone. All other dynamic tests were also decreased significantly. These changes were accompanied by increased RV and FRC and slightly decreased TLC.

Kagawa and Toyama [37] have reported on the results of limited studies involving four normal male subjects exercising while exposed to ozone at 1760 $\mu g/m^3$ (0.9 ppm) for 5 min. A significant decrease in SGaw was found during exposure and after 5 min of recovery.

Von Nieding et al.[59] studied the effects of 196 $\mu g/m^3$ (0.1 ppm) of ozone for 2 hr in 12 healthy males aged 24 to 38 years. Arterial oxygen tension (Pa$_{O_2}$) decreased significantly by 7 mm Hg from a mean of 84.6 ± 1.3 mm Hg to 77.6 ± 1.9 ($p < 0.01$) and returned to the initial level at the end of the 1-hr post-exposure period (84.3 ± 1.8 mm Hg). The alveolar-arterial gradient (AaD$_{O_2}$) increased with decreasing Pa$_{O_2}$ ($p < 0.01$). Airway resistance was significantly higher at the end of the 2-hr exposure than at the start. The subjects' airway resistance had not returned to normal 1 hr after exposure. Thoracic gas volume (TGV) did not change in the course of the experiment. Limitations in this study include the use of nonstandard measurement of flow resistance (Rt) and the use of arterialized capillary blood for P$_{O_2}$ measurement. Hence, until confirmed, these studies must be interpreted with caution.

Several investigators have reported apparent adaptation to pulmonary effects of ozone on repeated or chronic exposure. A discussion of these studies, along with a recent controlled exposure experiment, has been reported by Hackney et al.[29] Six men with respiratory hyperreactivity were exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone for 2 hr each day during 4 successive days. One subject showed little measurable response. The other five subjects showed decrements in lung function (generally on exposure days 1 to 3) that were largely reversed on day 4. These results suggest that some humans do not continue to experience the same decrements in lung function after repeated exposures to ozone. It is not known whether adaptation to other adverse effects of ozone exposure occurs.

Hackney and associates[27] also compared the response to ozone exposure of four southern Californians with that of four Canadians, whose cumulative ambient ozone exposure appeared to have been less than that of the Californians. In Hackney's southern California laboratory, all eight subjects underwent 2 hr of exposure to 730 $\mu g/m^3$ (0.37 ppm) ozone and intermittent light exercise. The investigators observed no statistically significant changes in lung function. However, the Canadians showed discernible post-exposure decrements in most lung function parameters measured, whereas the Californians did not. The Canadians also showed statistically significant changes in *in vitro* erythrocyte fragility, erythrocyte acetylcholinesterase activity, and serum vitamin E level. Among Californians, erythrocyte acetylcholinesterase activity was the only blood parameter showing post-exposure changes. To the investigators, adaptation of southern Californians to the effects of ozone exposure was the most plausible explanation for the observed results. However, selective migration of ozone-sensitive people away from southern California could not be wholly ruled out.

In a similar 4-day, 2-hr exposure protocol, Moller et al.[52] exposed six subjects to 730 $\mu g/m^3$ (0.37 ppm) ozone. Although conventional tests did not reveal any functional abnormalities, more sensitive nitrogen-clearance measurements showed that in hyper-reactive subjects, the specific distribution of ventilation had been altered.

Linn et al.[46] studied the effects of ozone exposure on symptomatology, respiratory physiology, and blood biochemistry in 22 physician-diagnosed asthmatic subjects (20 males and two females) living in the Los Angeles area. Persons with marked respiratory disability were excluded from study. Study participants varied in age from 19 to 59 years and covered a clinical range from slight wheezing to marked abnormality in forced expiratory performance. All but six used medication regulary.

Each subject was studied at the same time of day on 3 successive days in the Rancho Los Amigos exposure chamber. At the beginning of each day's study, baseline lung function measurements were obtained with subjects breathing purified air. Following this, subjects exercised lightly and rested over alternate 15-min periods for 2 hr. Exercise work loads were chosen to double each subject's resting minute ventilation (light exercise). In all studies, temperature and relative humidity were controlled to 31 ± 1°C and 35 ± 4 percent, respectively. Subjects were instructed to take oral medications on their regular schedules but to refrain from using inhaled bronchodilators during each 3-day study period.

On the first day, subjects were exposed only to purified air. On the second day, they were exposed to just enough ozone to permit detection of its odor at the beginning of the exercise-rest period, and to purified air for the rest of the period. On the third day, they were exposed to 0.20 to 0.25 ppm ozone throughout the period. At the end of each day's study period, the lung function tests of the subjects were repeated, their symptoms were assessed, and they were bled for measurement of hematologic and biochemical parameters.

To quantitate the effect of the 3-day study protocol on the parameters measured, the investigators repeated the entire protocol, with no ozone exposure, using a subsample of 14 persons (12 men and the 2 women) from the original group of subjects. On the average, subjects underwent the 3-day control study 10 months after the original study (range 1 to 23 months).

In all data analyses, the mean results of the whole group or the subsample were compared across different days of study. No formal attempt to identify or characterize unusually sensitive individuals was reported.

In the whole group of 22 subjects, the within-day difference between pre- and post-exposure lung function measurements differed significantly between the oxidant-exposure day and any other day for only one of nine measured parameters. This was total lung capacity (TLC), whose mean value increased by 0.06 liter on the odor-sham day and decreased by 0.10 liter on the oxidant-exposure day ($p < 0.05$). In the subsample of 14 subjects, no statistically significant differences in lung function between the ozone-exposure day and any other day of the first 3-day protocol was observed. For this group, between-day differences in lung function were slightly larger during the second 3-day (control) protocol than in the first.

For the whole group, the mean symptom score was higher (but not significantly so) on the ozone-exposure day than on the other 2 days, and it was virtually identical on the other 2 days. For the subsample in the first 3-day (exposure) protocol, the mean symptom score was also highest by a small margin on the ozone-exposure day. However, the highest daily score in the subsample occurred during the 3-day control protocol.

Ozone exposure was more strongly associated with blood biochemical changes than with symptoms or lung function changes. For the whole group, mean values of the following parameters differed significantly ($p < 0.05$) between the ozone-exposure day and at least one of the other 2 days (directions of difference on the ozone-exposure day are given in parentheses): total hemoglobin (lower), red cell fragility in H_2O_2 (higher), reduced glutathione (higher than odor-sham days, lower than clean-air days), acetylcholinesterase (lower), glucose-6-phosphate dehydrogenase (higher), and lactate dehydrogenase (higher). In the subsample of subjects in the 3-day exposure protocol, the same types of significant differences between ozone-exposure days and other days were observed, with the exception that no ozone-related difference in reduced glutathione occurred. In the 3-day control protocol, the only biochemical parameter showing significant differences between days was acetylcholinesterase.

The results of this study suggest that, in a reasonably representative sample of adult asthmatics, short-term exposure to realistic ozone levels (0.20 to 0.25 ppm) produces no measurable adverse effect on lung function. On the other hand, the results suggest a moderate tendency toward increased symptom frequency in asthmatics at the ozone level used, and they strongly suggest that ozone at this level can alter certain biochemical and hematologic parameters.

The changes that Linn et al.[46] have associated most confidently with ozone exposure are changes of the least certain clinical significance. Since most of the observed biochemical and hematologic effects relate directly or indirectly to the oxygen-carrying capacity of the blood, they might be unusually important in groups, such as asthmatics, with compromised pulmonary function. Also, as the authors mention, these findings raise the possibility that asthmatics may react biochemically at lower ozone concentrations than do normal persons. However, the observed changes were small when compared to the normal inter-individual variability of the parameters measured, and they were considerably smaller than would occur with obvious clinical disease. Thus the question as to whether these changes represent harmful effects in asthmatics remains open.

As mentioned, Linn et al.[46] reported only group mean results. Thus their findings do not touch on the question of whether certain individual asthmatics may be particularly sensitive or resistant to ozone concentrations of 0.20 to 0.25 ppm. Finally, their findings do not allow inference as to what the effects of ozone might be in childhood asthma.

The effects of sequential exposures separated by 1 day to 2 months, in combination with different concentrations of ozone, have been investigated by Hazucha et al.[34] The 2-hr ozone exposure protocol for 16 subjects, divided into four groups, was as follows:

1. Group A: 780 $\mu g/m^3$ (0.4 ppm), 1- to 2-month delay, 390 $\mu g/m^3$ (0.2 ppm), 1-day delay, 780 $\mu g/m^3$ (0.4 ppm) ozone;
2. Group B: 390 $\mu g/m^3$ (0.2 ppm), 1-day delay, 780 $\mu g/m^3$ (0.4 ppm), 1- to 2-month delay, 780 $\mu g/m^3$ (0.4 ppm);
3. Group C: 1180 $\mu g/m^3$ (0.6 ppm), 1- to 2-month delay, 390 $\mu g/m^3$ (0.2 ppm), 3-day delay, 1180 $\mu g/m^3$ (0.6 ppm);
4. Group D: 1180 $\mu g/m^3$ (0.6 ppm), 1- to 2-month delay, 780 $\mu g/m^3$ (0.4 ppm), 3-day delay, 1180 $\mu g/m^3$ (0.6 ppm).

From all four groups, only group D showed clear signs of adaptation (i.e., response to the third challenge exposure was less marked than a response observed earlier after exposure to the same concentration). Although the size of the groups was small, the results obtained indicate that the extent of changes induced by the challenge exposure depend primarily on the ozone concentration during pre-exposures and the time interval between preconditioning and challenge exposures.

In most reported studies,[3,12,16,26,32,35,45,56] an association between symptoms and changes in lung function was usually found. Ozone-induced defects in function were not usually found in the absence of definite symptoms of ozone-induced respiratory irritation.

The newer experimental studies described above[2,12,17,19,32,35,37,39,56] show that significant adverse changes in lung function occur in humans at ozone concentrations of 725 $\mu g/m^3$ (0.37 ppm) and higher. Some limited studies show evidence of human health effects of exposure to pure ozone at concentrations as low as 490 $\mu g/m^3$ (0.25 ppm) (see Figure 9-3).[28,32] The prelimininary findings of

Von Nieding et al.[59] suggest decrements in lung function at 196 $\mu g/m^3$ (0.1 ppm) exposure for 2 hr.

Hematology

Although the toxic effects of oxygen on red blood cells (RBC) have been known for a long time, it was not until recently that injurious effects of a much stronger oxidant, ozone, have been investigated. Initial studies concerning ozone toxicity on RBC in animals showed that ozone and oxygen share a great number of pharmacological and toxicological features. The striking similarity between the toxicity of these two gases has attracted considerable interest among both researchers and clinicians and an increasing number of articles, including a review,[48] have been published on this problem.

IN VITRO STUDIES

The RBC constituent most sensitive to ozone damage seems to be the membrane. The increased breakdown of unsaturated fatty acids in the red cell membrane was observed after exposure to 780 $\mu g/m^3$ (0.4 ppm) ozone for 2 hr. The degree of unsaturation of the acids was positively correlated with the extent of lipid peroxidation.[1] The same intensity of exposure significantly reduced acetylcholinesterase activity and decreased neuraminic acid levels in cell membranes. Subsequent incubation of the exposed cells in plasma resulted in complement-mediated RBC membrane damage as measured by acid, sucrose, and insulin hemolysis tests. Since such a reaction is characteristic of RBC in paroxysmal nocturnal hemoglobinuria (PNH) disorder, Goldstein et al. hypothesized that the ozone-induced changes in the red cell membrane have produced PNH-like RBC's.[21,22] In addition, a recent study from the same laboratory showed that exposure of RBC under similar experimental conditions also caused a loss of native protein (tryptophan) fluorescence in the cell membrane.[21] With regard to the above studies, it can be argued that the concentration of ozone used was unrealistically high, but serious disturbances in cell chemistry caused by such concentrations can be detected much more easily than the subtle changes induced by lower levels. Increased sophistication and sensitivity of various tests have allowed considerable reduction of ozone concentrations in hemotological studies. Recently, Kindya and Chan [42] demonstrated that 3-min exposure of 1.0- ml sample of erythrocyte membrane fragments to 12 mmol ozone significantly decreased ouabaine-sensitive adenosine

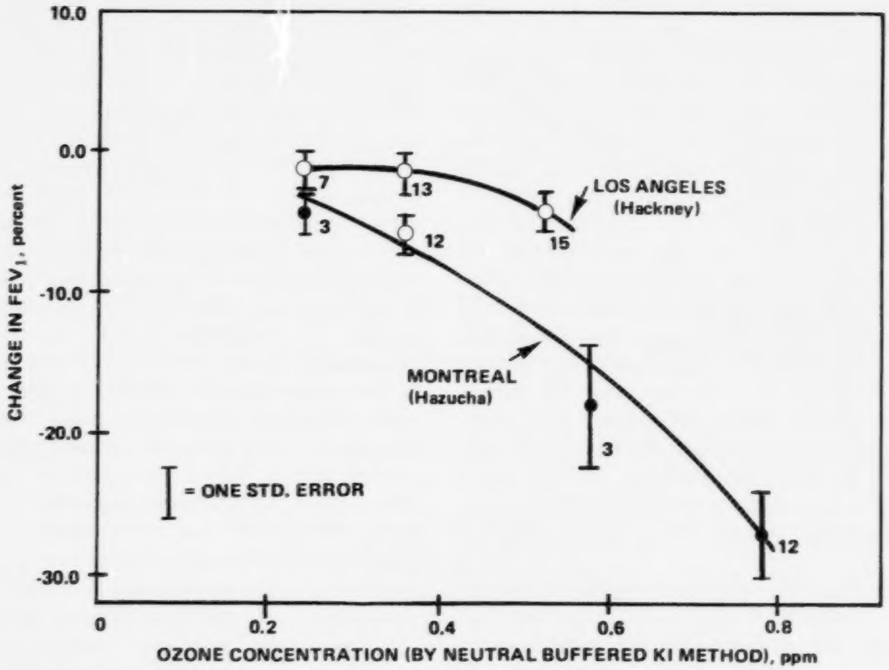


Figure 9-3. Dose-response curves for Los Angeles and Montreal subjects.[32,28]

triphosphatase (ATPase) activity. Since this enzyme plays a major role in maintaining intactness of erythrocytes, the investigators postulated that such inactivation of the activated ATPase in the cell membrane may lead to increased osmotic fragility and sphering of RBC. It is of interest to note that both cysteine and ascorbic acid, when added to the red cell membrane fragments before exposure, afforded protection to the ATPase against ozone.

In recent years, a number of compounds have been studied as to their antioxidant effects on various enzyme systems. Goldstein and Levine[23] demonstrated that *p*-aminobenzoic acid inhibited the ozone-induced loss of activity of acetycholinesterase in red cell membranes. Menzel et al.[50] showed that addition of vitamin E (α-tocopherol) or d-α-tocopheryl acetate prevented formation of Heinz bodies formed in RBC by ozonides, which are potent indicators of lipid peroxidation in cellular membranes.[49]

IN VIVO STUDIES

Brinkman and Lamberts[7] were the first to report that inhalation of ozone interfered with oxygen exchange in the capillaries of skin. In a later study, Brinkman et al.[8] demonstrated potentiation of erythrocyte sphering in subjects exposed to 490 μg/m^3 (0.25 ppm) ozone for periods of 30 to 60 min. More than 10 years later, Buckley et al.[9] employed a comprehensive battery of biochemical tests to investigate the toxic effect of ozone to RBC of subjects exposed to 980 μg/m^3 (0.5 ppm) ozone for 2¾ hr. The observed changes were similar to the effects reported previously in either animal or human blood *in vitro* studies. They demonstrated a significant increase in RBC fragility. Lactate dehydrogenase (LDH) and glucose-6-phosphate dehydrogenase (G-6-PD) enzyme activities were increased, while acetylcholinesterase activity and reduced glutathione (GSH) were depressed. Cell membrane glutathione reductase (GSSRase) showed no change in activities, whereas serum GSSRase activities were decreased. The post-exposure level of serum vitamin E was significantly higher as was lipid peroxidation. Clearly these results indicate that ozone or its by-products can induce changes across the alveolo-capillary barrier.

Although the presented data display a spectrum of ozone interference with biochemical mechanisms, the physiological significance of all these studies remains to be established. To evaluate clinical implications of the ozone-induced cellular damage, quantitative studies at realistic concentrations are needed.

Mutagenesis

The work described in the section on chromosomes in Chapter 8 of this document prompted investigators to evaluate the potential for ozone to cause chromosomal aberrations. Merz et al.[51] reported results from six men exposed to 980 μg/m^3 (0.5 ppm) ozone, two of them for 6 hr and the other four for 10 hr. Blood was obtained from the first two subjects before and immediately after exposure. The other four subjects had blood drawn before exposure, immediately after, and then at 2 and 6 weeks following exposure. The lymphocytes were cultured and prepared for chromosomal analysis following standard procedures. No true chromosomal-type abnormalities were observed except for one instance in one individual. However, there were lesions that the authors interpreted as achromatic, unrepaired single-strand breaks, and chromatid deletions. Although such aberrations were seen in some of the pre-exposure preparations, the authors reported an increased frequency of abnormalities 2 weeks post-exposure, with reversion to lower frequency 6 weeks post-exposure.

Because of the observations of Merz et al.,[51] EPA conducted a study to evaluate the potential human mutagenic effects of ozone in a more rigorous fashion. Thirty nonsmoking, normal, young male volunteers breathed ozone at 780 μg/m^3 (0.4 ppm) during 4 hr in a controlled-environment laboratory.[58] The cytogenic results of this study have been reported by McKenzie et al.[47] Blood was drawn from the subjects before exposure, immediately following exposure, and at 3 days, 2 weeks, and 4 weeks after exposure. Lymphocytes were cultured for 48 hr, and slides were prepared in the usual way, with 100 metaphase spreads per subject per treatment scored for chromosomal aberrations. A total of 13,000 human lymphocytes were cytogenetically analyzed in this study. Cells with suspected aberrations were photographed, destained, restained with banding procedure, and photographed again to identify the specific chromosomes and regions involved. The data from this experiment consist essentially of five measurements on each subject over the period of 1 month. Thus the appropriate statistical analysis of variance for reported measurements was done on the raw data and on appropriately arcsin-transformed observations. There was no significant difference in any of the observations for the respective blood sampling times. The authors conclude that there is no detectable human cytogenetic effect related to exposure to ozone under the conditions of this experiment.

These findings are in contrast to those reported by other investigators. The *in vitro* and animal studies discussed in the section on chromosomes in Chapter 8 of this document used either extremely high ozone doses or inadequate controls. The study of Merz et al.[51] used higher ozone concentrations and much longer exposures than that of McKenzie et al.[47] Also, the small number of subjects in the Merz et al. study did not permit the rigorous statistical analysis of the McKenzie et al. study. Clearly, additional evaluation of the human mutagenic potential of ozone is needed, and such research is now in progress. Evidence now available, however, fails to demonstrate any mutagenic effect of ozone in humans when exposure schedules are used that are representative of such exposures of the population at large as might actually occur in urban areas.

Controlled Studies of Human Health Effects of Peroxyacetylnitrate

Experiments conducted on a group of male college students averaging 21 years of age have suggested that exposure to PAN results in increased oxygen uptake during exercise compared to uptake during breathing of clean air.[57] This report, however, did not adequately describe the experimental design or the statistical analysis. Subjects were exposed to 1485 $\mu g/m^3$ (0.3 ppm) PAN by breathing through the mouth (nose clamps were used) for 5 min while at rest. Then the subjects were immediately engaged in 5 min of exercise on a bicycle ergometer. Both air containing PAN and air free of PAN were used. Expiration velocity was reduced after exercise. The changes could reflect an increase in the effort needed in breathing as a result of the exercise or an increase in airway resistance.

In a series of studies of the effects of PAN and carbon monoxide on healthy young and middle-aged males during treadmill work in an exposure chamber, Raven and coworkers found no changes in maximum aerobic power when the subjects were exposed to 1337 $\mu g/m^3$ (0.27 ppm) PAN.[13,53,54,58,59,60] When the subjects worked at a level requiring 35 percent of maximum oxygen consumption for 3.5 out of 4 hr of continuous exposure to 1188 $\mu g/m^3$ (0.24 ppm) PAN, no significant changes were observed in the metabolic, cardiovascular, or thermoregulatory responses.[18] A small (4 to 7 percent) but significant,[18] reduction in standing FVC was observed in the younger group of subjects.[18]

SUMMARY

Convincing new information on the health effects of oxidant exposure has emerged from controlled studies on humans, from which tentative dose-response curves have been constructed. The new data show statistically significant reduced pulmonary function in healthy smokers and nonsmokers at ozone concentrations at and above 730 $\mu g/m^3$ (0.37 ppm) for 2-hr exposures. However, a recent study by Von Nieding et al.[59] showed some effect at as low a concentration of ozone as 0.1 ppm for 2 hr. Some studies suggest that mixtures of sulfur dioxide and ozone at a concentration of 0.37 ppm are more active physiologically than would be expected from the behavior of the gases acting separately. Wide variation in response among different individuals is a general finding in studies of oxidants, as well as other pollutants. Undesirable health effects of oxidant air pollution exposure are increased by exercise and, as judged by informal surveys, many people apparently limit strenuous exercise voluntarily when oxidant pollution is high.

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10. EPIDEMIOLOGIC APPRAISAL OF PHOTOCHEMICAL OXIDANTS

INTRODUCTION

A review is presented in this chapter of the evidence gathered to date in the epidemiologic studies of whether or not ozone and other photochemical substances exert deleterious effects on human health. Every effort has been made to describe study designs and investigative methods thoroughly so that readers with either a technical or nontechnical background may judge both the reliability of findings presented and the degree to which observed effects may be attributed specifically to photochemical air pollution.

This chapter consists of four sections. The first deals with effects of short-term (generally less than 1 week) exposures to photochemical air pollutants. In many studies of short-term exposures, pollutant concentrations have been measured on the same day, or even in the same hour, as the health indices to which they have been compared. The second section deals with effects of long-term exposures to photochemical air pollutants. The health data for most of these studies have been collected at one point in time. Thus, even though certain health impairments have been associated with long-term pollutant exposures, it has generally not been possible to state the duration of exposure that may have produced those impairments.

The sections of this chapter dealing with short-term and long-term exposures have been divided into subsections in which studies of similar effects are reviewed. Following each subsection is a discussion that assesses the reliability of current evidence and identifies the areas of uncertainty requiring future research. Experimental studies, which give some indication of physiological mechanisms underlying findings of epidemiologic studies, are discussed where practicable. The degree to which observed epidemiologic findings may be attributed specifically to photochemical air pollution or to individual components of this broad class is also assessed. Finally, an effort is made to identify specific pollutant concentrations and the duration of exposure necessary to promote the observed effects.

Following the sections on short-term and long-term exposures, a brief assessment of the attitudes of laymen and physicians toward oxidant pollution will be presented. Finally, the available epidemiologic evidence related to oxidant pollution will be summarized.

EFFECTS OF SHORT-TERM PHOTOCHEMICAL OXIDANT EXPOSURES

To identify the acute health effects of photochemical oxidant pollution, observations of the same populations of communities are made during periods of both high- and low-level pollution. Changes in health-related indices are compared with short-term pollutant concentrations measured as 24-hr averages, hourly maxima, or instantaneous peak concentrations.

Daily Mortality in Relation to Variations in Oxidant Levels

MORTALITY AMONG RESIDENTS AGED 65 YEARS AND OVER

Over a 3-year period from 1954 to 1957, the California State Health Department conducted a study of the relationship between daily concentrations of photochemical oxidants and daily mortality among residents of Los Angeles County ages 65 years and over.[6,7,8] During these years, photochemical oxidant concentrations in Los Angeles County were generally measured by the potassium iodide (KI) method. Ozone concentrations were generally measured by visual assessment of rubber cracking. Both methods were calibrated with unbuffered 2-percent KI solution. The number of deaths per day was related to two indices—maximum daily temperature at the downtown weather bureau, and oxidant concentrations from August through November 1954 and from July through November 1955.

Data collected over these periods of time are summarized in Table 10-1 and Figure 10-1. Examination of the table and figure reveals a pronounced effect of the September 1955 heat wave on mortality. The figure reveals no consistent association between mortality and high oxidant concentrations in the absence of unusually high temperatures.

TABLE 10-1. AVERAGE NUMBER OF DEATHS PER DAY RESULTING FROM CARDIAC AND RESPIRATORY CAUSES AMONG RESIDENTS OF LOS ANGELES COUNTY, AGED 65 AND OVER, AS RELATED TO OXIDANT CONCENTRATIONS AND MAXIMUM DAILY TEMPERATURE BY MONTH, 1954-55

	Totals		Temperature readings: 10°-15°C (50°-59°F)		16°-20°C (60°-69°F)		21°-26°C (70°-79°F)		27°-31°C (80°-89°F)		32°-37°C (90°-99°F)		>38°C (>100°F)	
Concentration, ppm	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths	Number of days	Average number of deaths
August 1954														
Low (00-0.24)	8	29.9					4	28.2	4	31.5				
Medium (0.25-0.49)	22	32.2					7	30.1	12	31.8	3	39.0		
High (0.50+)	1	32.0					1	32.0						
September 1954														
Low (00-0.24)	1	26.0					1	26.0						
Medium (0.25-0.49)	25	32.1					5	29.6	16	35.1	4	36.0		
High (0.50+)	4	33.3							4	33.3				
October 1954														
Low (00-0.24)	9	39.4			1	31.0	6	38.2	2	47.5				
Medium (0.25-0.49)	14	37.1			1	43.0	10	36.0	2	39.5	1	37.0		
High (0.50+)	8	36.3					3	36.7	5	36.0				
November 1954														
Low (00-0.24)	16	38.6	1	39.0	5	36.0	4	42.0	6	38.6				
Medium (0.25-0.49)	14	37.9			4	36.8	5	34.8	5	41.8				
High (0.50+)														
July 1955														
Low (00-0.24)	4	35.0					1	28.0	3	37.3				
Medium (0.25-0.49)	11	34.8					5	34.4	6	35.2				
High (0.50+)	2	42.0					1	42.0	1	42.0				
No readings	14	37.5					10	38.7	4	34.5				
August 1955														
Low (00-0.24)	5	36.8							4	35.5			1	42.0
Medium (0.25-0.49)	16	38.4							16	38.4				
High (0.50+)	4	37.2							4	37.2				
No readings	6	37.8							4	38.5	2	36.5		
September 1955														
Low (00-0.24)	5	29.0			1	33.0	3	27.3	1	30.0				
Medium (0.25-0.49)	13	61.5					4	30.8	3	43.7	1	52.0	5	98.8
High (0.50+)	7	50.3					1	32.0	1	28.0	3	40.7	2	85.0
No readings	5	34.8					4	33.2			1	41.0		
Mean														
Low (00-0.24)	48	U33.5[a] W35.5	1	39.0	7	33.3	19	31.6	20	36.7			1	42.0
Medium (0.25-0.49)	115	U39.1 W37.9			5	39.9	36	32.6	60	37.9	9	41.0	5	98.8
High (0.50+)	26	U38.5 W40.0					6	35.7	15	35.3	3	40.7	2	85.0
No readings	25	U36.7 W37.0					14	36.0	8	36.5	3	38.8		
Total days			1		12		75		103		15		8	
Total deaths														
Unweighted				39.0		36.6		34.0		36.6		40.2		75.3
Weighted				39.0		36.1		33.2		37.2		40.5		88.3

[a] U = unweighted, W = weighted.

Table 10-1 contains too few data to yield any conclusive trends. However, examination of the table's marginal means suggests that the effect of temperature on death rates, at least during the months considered, is smallest in the comfortable temperature range of 21° to 26°C (70° to 79°F). Such examination also suggests a positive association between oxidant exposure and mortality in this temperature range. Examination in Table 10-1 of overall mean death rates by pollution exposure category also suggests such an association.

It must be emphasized that Table 10-1 does not reveal a convincing relationship between oxidant exposure and daily mortality. Indeed, the table contains far too few data to warrant conclusions of any kind, except perhaps that the effect of unseasonable temperatures on mortality is large enough to obscure any effect that oxidant pollution may have. However, Table 10-1 also arouses interest in the hypothesis that any effect that oxidant might exert on mortality may be most apparent at times when temperatures are seasonal and comfortable. As yet, this hypothesis has not been adequately tested.

MORTALITY AND HEAT WAVES

In the Los Angeles Basin, high temperatures and elevated oxidant concentrations tend to occur simultaneously. The question thus arises whether oxidant exposure augments the effect of temperature on mortality rates. Oechsli and Buechley[41] considered this question briefly in a study of the effect on mortality of three Los Angeles heat waves occurring in 1939, 1955, and 1963. Daily mortality during these heat waves was compared with daily mortality occurring immediately before and after each heat wave and with mortality during the same season in 1947, when no heat wave occurred.

Statistically significant increases in mortality rates were observed during each heat wave, particularly among elderly persons. However, there was no apparent difference between the effects of the 1939 and 1955 heat waves on mortality rates. Quite probably, considerably less photochemical oxidant pollution accompanied the 1939 heat wave than that of 1955. (Oxidant concentrations were not routinely measured in 1939.) The comparison of the two heat waves thus

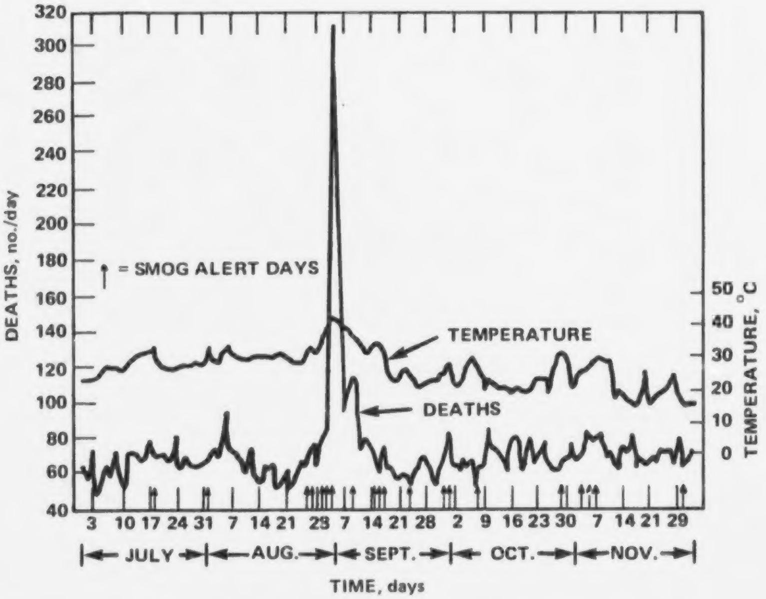


Figure 10-1. Comparison of deaths of persons aged 65 years and over, and maximum daily temperatures, Los Angeles County, July 1 to November 30, 1955.[6,7,8]

suggests that high photochemical oxidant concentrations do not notably augment the effect of high temperatures on mortality. On the other hand, it must be remembered that Oechsli and Buechley did not statistically determine whether a relationship between mortality and oxidant exposure existed.

MORTALITY OF NURSING HOME RESIDENTS

The California State Health Department also made an effort to determine whether patients in Los Angeles nursing homes, many of whom are chronically ill, experienced increased mortality during or just after days of high oxidant concentrations.[6,7,8] Deaths and transfer to hospitals among residents of 16 Los Angeles nursing homes having a total of 358 beds were recorded for 1954. An unusually large number of patients died following a particularly heavy episode of smog during 1 week of the study period. During the same week, however, the number of residents transferred to hospitals did not appear to be unusually high. Neither the number of deaths nor the number of transfers to hospitals was elevated during a high-smog period about a month earlier than the one mentioned above. Measurements of pollutant concentrations were not reported.

A larger study of the nursing home population was conducted from July through December 1955, during which all nursing homes in Los Angeles County containing 25 or more beds were surveyed. Daily mortality rates were reported from 92 homes with a total of 3826 beds. Daily mortality, the corresponding maximum daily temperature, and the occurrence of smog-alert days, with ozone concentrations of 590 $\mu g/m^3$ (0.30 ppm) or higher, are shown in Figure 10-2. The heat wave in late August and early September, during which several smog-alert days occurred, showed a striking effect on mortality. At no other time could a relationship between daily mortality and smog-alert days be discerned.

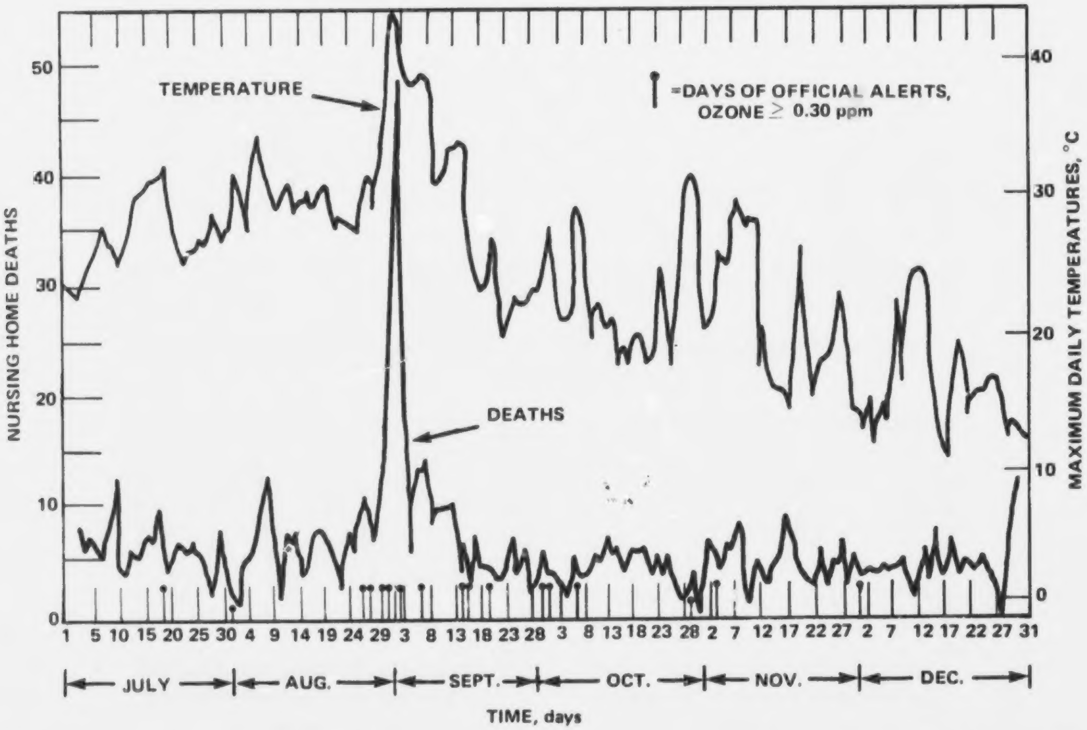


Figure 10-2. Comparison of nursing home deaths, maximum daily temperature, and smog-alert days in Los Angeles County, July through December 1955.[6,7,8]

TWO-COMMUNITY STUDY

Massey et al.[33] compared daily mortality in two areas of Los Angeles County selected to be similar in temperature but different in air pollution levels. The investigators constructed two synthetic communities, one of intermediate pollution and one of high pollution, containing a combined population of 944,391 persons. The pollutant variables used in data analysis were the daily maximum and mean oxidant levels, as measured by the potassium iodide (KI) method and sulfur dioxide and carbon monoxide concentrations. Intercommunity differences in temperature were also considered. The mean number of daily deaths in the intermediate pollution area was subtracted from the mean number of deaths in the high pollution area, and the differences were related by multiple correlation and regression to differences in pollution concentrations. No significant correlations between differences in mortality and differences in pollutant levels were observed. Mean daily death rates per 100,000 were 3.18 and 3.06, respectively, in the intermediate- and high-pollution communities.

MORTALITY RESULTING FROM CARDIAC AND RESPIRATORY DISEASES

Hechter and Goldsmith[22] analyzed the effect of pollutant concentrations on average daily mortality from cardiac and respiratory diseases in Los Angeles County for the years 1956 through 1958. During these years, photochemical oxidant concentrations were measured in Los Angeles County by the KI method, using an unbuffered 2-percent KI solution as the calibration reagent. Daily mortality, (averaged within each month of the study) fluctuated between 1.0 and 1.3 per 100,000 population. These fluctuations were approximately 180 degrees (6 months) out of phase with fluctuations in oxidant and temperaure values and approximately in phase with maximum carbon monoxide concentrations (Figure 10-3). To remove the effect of season of year, the authors fitted Fourier curves to the data. The residual variations from these fitted curves for each of the variables were presumed to be independent of season. The relationship between pollution or temperature on one day with the value of the same variable on the preceding or following days was also accounted for

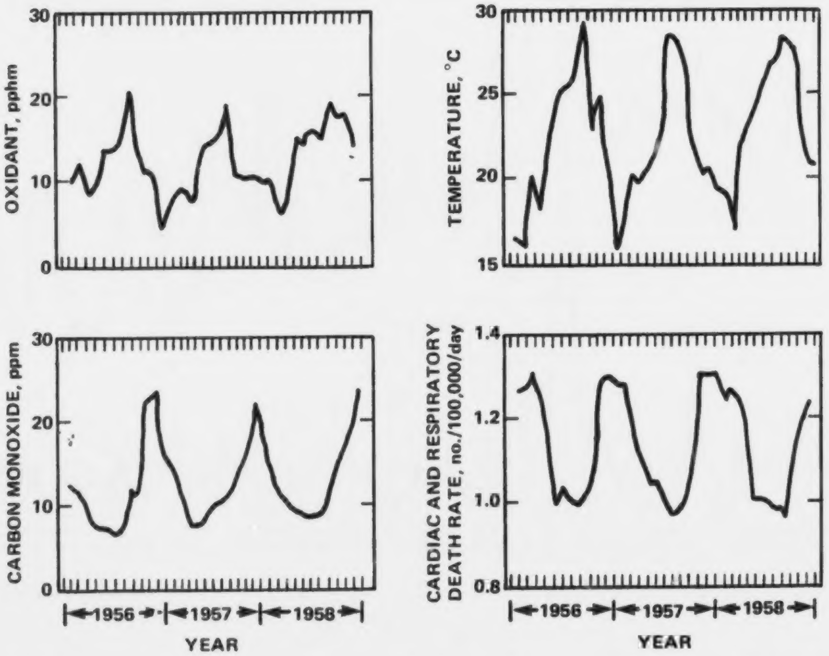


Figure 10-3. Comparison of maximum concentrations of oxidant and carbon monoxide, maximum temperature, and daily death rate for cardiac and respiratory causes, Los Angeles County, 1956-58.[22]

in the analysis. When residuals from the fitted curves were thus analyzed, no significant correlations between pollutants and mortality for cardiorespiratory diseases were found. Neither were there significant correlations when a 1- to 4-day lag between exposure and mortality was applied.

Mills[36] correlated daily cardiac and respiratory mortality in Los Angeles with corresponding daily maximum oxidant concentrations for the following periods: 1947 through 1949; August 17, 1953, through December 31, 1954; and January 1 through September 30, 1955, excluding the severe heat wave of September 1 through 4. To correct for possible seasonal effects on mortality, Mills compared mean daily death rates on low-smog days to mean rates on higher-smog days occurring within the same month.

For 1947-49, mortality data were related to the Stanford Research Institute's smog index,[56,57,58] which was based on meteorologic conditions, not on air pollution measurements. (Coefficients of correlation between the smog index and maximum ozone measurements, supplied by the U. S. Rubber Co., ranged from 0.08 to 0.949. All were statistically significant at $\alpha = 0.05$.) For 1953-55, mortality data appear to have been related to daily maximum total oxidant measurements supplied by Dr. A. J. Haagen-Smit of the California Institute of Technology. These measurements were presumably made with the phenolphthalein method.

In all periods, Mills[36] observed a consistent positive relationship between daily mortality and daily maximum ozone or oxidant concentration. This relationship held for persons under age 65 as well as for older persons. On the average, daily death rates in 1947-49 were about 7 percent higher when the smog index was six through nine (corresponding to maximum ozone concentrations of about 0.36 to 0.37 ppm) than when the smog index was zero through five. In the same period, daily death rates when the smog index was 10 or above (corresponding to maximum ozone concentrations of at least 0.42 ppm) averaged 12 percent higher than when the smog index was zero through five. For 1953-55, the mean daily death rate when maximum total oxidant exceeded 0.2 ppm averaged about 5 percent higher then when maximum total oxidant was below 0.2 ppm. Mills,[36] like the California Department of Health,[6,7,8] performed an analysis of death rates in Los Angeles nursing homes during the second half of 1955. Unlike the California Department of Health, Mills applied a seasonal correction factor to death rates within each month of this period. Mills' analysis suggested a continuous increase in daily death rates with increasing daily maximum oxidant concentrations above 0.2 ppm. Corrected daily death rates ranged from 4.72 when daily maximum oxidant was below 0.2 ppm, to 6.23 when maximum oxidant was 0.8 ppm or above. The correlation between seasonally corrected death rate and maximum oxidant was 0.206 ($p = 0.01$).

In a separate report, Mills[35] stated that more than 334 deaths in Los Angeles during 1954 were associated with daily oxidant or ozone maxima above 0.2 ppm.

The California Department of Health has specifically questioned Mills' findings. In *Clean Air For California*,[6,7,8] the Department explains that the Stanford Research Institute's smog index, by which Mills classified mortality data for 1947-49, was developed as a prediction device and not as a measurement of air pollution. The Department also states that the abandonment of the smog index by the Institute indicated that it was not satisfactory in either respect. They add that Mills' manner of selecting smog-free days to be used for comparison may not have been entirely satisfactory.

The California Department of Health also applied the essential details of Mills' analytical procedure to Los Angeles County cardiac and respirator death rates during August-November 1954.[6,7,8] Results of the California analysis are shown in Table 10-2. Comparison of mortality rates on smoggy and smog-free days within each of the months shown reveals no effect of oxidant pollution on mortality. Thus, as stated in references,[6,7,8] and the Breslow and Goldsmith's report,[4] analyses performed in California did not support Mills' conclusion that 334 deaths in Los Angeles during 1954 were associated with oxidant levels above 0.2 ppm.

Interpretation of the results summarized in Table 10-2 is clouded because references[6,7,8] do not contain an explanation of criteria used to differentiate smoggy from smog-free days. Also, the temperatures on the two types of days cannot be ascertained from three references. However, that Mills and the California Department of Health have interpreted Los Angeles mortality data differently is undeniable.

DISCUSSION OF MORTALITY STUDIES

As yet, no convincing association between daily mortality and daily oxidant concentrations in Los Angeles has been shown. Mills has certainly described a positive association between mortality and oxidants. However, because the smog index used for much of his analysis appears to have been inaccurate, and because investigators in California have observed no oxidant-mortality association in data used by Mills himself, the vigor with which Mills' conclusions can be advanced must be considerably restricted. However, careful examination of available studies raises the interesting question as to whether oxidants might affect mortality at times when temperatures are comfortable and seasonal. To date, most efforts to determine whether oxidants affect mortality have focused on high-smog periods. During such periods, temperatures also tend to be high and may thus obscure or eliminate any effect of oxidants on mortality. The data in Table 10-1, which are apparently the only extant cross-tabulation of mortality by pollution and temperature, suggest that the effect of oxidant on mortality may be strongest or most readily detectable, when temperatures are seasonal. Indeed, it is plausible that oxidants would noticeably affect mortality at no other times.

Thus whether oxidants affect daily mortality has not been conclusively resolved. Recent advances in statistical analysis of mortality data, coupled with the accumulation of such data over the two decades since most oxidant-mortality studies were performed in Los Angeles, should enable investigators to clarify this issue considerably over the next several years.

Hospital Admissions in Relation to Oxidant Levels

ADMISSIONS TO LOS ANGELES COUNTY HOSPITAL, 1954

The California State Health Department examined several categories of admissions to Los Angeles County Hospital from August through November 1954.[6,7,8] These categories were asthma, other respiratory conditions, and cardiac conditions. Total admissions, the number of persons with acute conditions in all units, and the number who died were tabulated. A formal statistical analysis does not appear in *clean-air-for-California reports.*[6,7,8] However, visual inspection of these data reveals no consistent relationship between admissions and high smog periods.

Brant and Hill[2,3] studied 9- to 90-year-old patients with respiratory or cardiovascular diagnoses who were admitted to or discharged from Los Angeles County Hospital between August and December 1954. Patients were considered only if they had resided for at least 3 years in an area within 13 km (8 miles) of downtown Los Angeles. Although the method of selecting patients for the study was not described, the selection process yielded 246 cardiovascular admissions and 122 respiratory admissions. Daily average total oxidant concentrations between 6 a.m. and 1 p.m., as measured about 1.6 km (1 mile) from the hospital, were the only indices of air pollution employed in statistical analyses. Multiple regression analyses were used to relate hospital admissions to atmospheric and meteorological variables. Calculations included regressions of hospital admissions as late as 4 weeks after the occurrence of a given set of environmental measurements, which included total oxidant, temperature, and relative humidity. Brant obtained a positive partial correlation coefficient of 0.982 between the total oxidant level and the number of respiratory and cardiovascular admissions occurring 4 weeks later. However, the correlations between total oxidants and admissions during the same week and 2 weeks later were, respectively, -0.986 and -0.870.

Although Brant's findings are interesting, they must be regarded as inconclusive for four reasons. First, it is difficult to rationalize why hospital admissions should be more dependent on oxidant

TABLE 10-2. COMPARISON OF RESPIRATORY[a] AND CARDIAC[b] DEATHS DURING SMOG AND SMOG-FREE PERIODS OCCURRING IN LOS ANGELES COUNTY, AUGUST-NOVEMBER 1954[6,7,8]

Smog periods	Total deaths, selected causes	Daily average	Smog-free periods	Total deaths, selected causes	Daily average
September 21-24	181	46	September 12-15	191	48
October 15-20	294	49	October 1-6	303	50
November 24-28	235	47	November 16-20	244	49

[a] Respiratory tuberculosis (001-008); pneumonia, all forms (490-493). Sixth revision, international list numbers.

[b] Diseases of the heart (410-443). Sixth revision, international list numbers.

levels 4 weeks before than on more recent levels. Second, the partial correlation coefficients between cardiovascular admissions and total oxidant concentrations during both the week of the admissions and 2 weeks before were very highly negative. Third, as far as we can determine, Brant did not examine the variables used in his statistical models for autocorrelation. Fourth, no indices of pollution other than oxidant concentrations were considered.

HOSPITAL ADMISSIONS THROUGHOUT THE CITY OF LOS ANGELES

In two studies, Sterling et al.[59,60] assessed the influence of air pollution exposures on persons admitted to hospitals with more than 100 beds in the city of Los Angeles. Only patients who were members of Blue Cross were considered. Discharge diagnoses were assigned to one of three categories—highly relevant, relevant, and irrelevant—which reflected the presumptive susceptibility of the diagnoses to air pollution exposure. Included in the "highly relevant" category were allergic disorders, inflammations of the eye, and acute upper and lower respiratory infections. Included in the "relevant" category were cardiovascular diseases and other respiratory diseases. All other conditions were included in the "irrelevent" category. The authors corrected both number of admissions and pollution exposure measurements for day-of-week effects. Nine pollution indices, including oxidant, ozone, carbon monoxide, sulfur dioxide, nitrogen dioxide, nitric oxide, and particulate matter, were considered in Sterling's analysis. Air pollution measurements were obtained from the Los Angeles County Air Pollution Control District. For each pollutant, the exposure index used in statistical analyses was the average of the maximum and minimum concentrations for each day of the study.

In Sterling's first study,[59] air pollution indices were correlated with the number of hospital admissions occurring on the same day in each diagnostic category. In all, 223 days between March and October 1961 were considered. Correlation coefficients between admissions for highly relevant diseases and all nine pollution indices measured were all statistically significant at $\alpha = 0.05$. All but one correlation (that with sulfur dioxide) were significant at $\alpha = 0.01$. The magnitude of these correlation coefficients ranged from 0.16 (for sulfur dioxide) to 0.25 (for particulate matter). Correlations between relevant diseases and pollution were significant for oxidant, ozone, and sulfur dioxide. For all relevant diseases, the highest correlations were observed with ozone (0.27), sulfur dioxide (0.27), and oxidant (0.25). For all pollutants except sulfur dioxide, correlations between irrelevent diseases and pollution were negative or not significant. Coefficients of correlation between temperature or relative humidity and admissions were not significant in all disease categories.

In the second study performed by Sterling et al.,[60] length of stay in the hospital was correlated with air pollution and meteorologic variables over the same time period, as in the first study. The absolute value of correlation coefficients was usually less than 0.1 and never exceeded 0.169. Despite their diminutive values, coefficients of correlation between length of stay and all pollution indices were statistically significant at $\alpha = 0.05$ for the irrelevent diseases and for all disease categories combined. All correlations between pollution indices and length of stay for irrelevant diseases and all disease groupings combined were positive, except that for ozone concentration. The only pollutants consistently and significantly positively correlated with length of stay for relevant disease categories were sulfur dioxide, nitrogen dioxide, and total oxides of nitrogen. Coefficients of correlation between humidity and temperature and length of stay in all disease categories were negative and significant at $\alpha = 0.01$. This finding with respect to temperature contrasts with the findings of several of the mortality studies cited above.

DISCUSSION OF STUDIES OF HOSPITAL ADMISSIONS

Studies of the relationship of short-term oxidant exposures to hospital admissions have yielded mixed results. Even studies in which a positive association between exposures and admissions has been observed must be considered inconclusive. As mentioned above, it is difficult to rationalize Brant's observation of a strong association between pollutant levels in 1 week and hospital admissions 4 weeks later, in the face of highly negative associations in the intervening weeks. Also, in the light of Sterling's studies, one must wonder whether Brant, if he had chosen to calculate them, would not have obtained correlation coefficients between admissions and pollutants other than oxidants that were comparable to those calculated for oxidants.

Thus even if the association between short-term exposures and hospital admissions observed in Los Angeles proves to be valid, available evidence in this field will not support the incrimination of certain pollutants and the exoneration of others. Also, available evidence gives no indication of pollutant concentrations at which hospital admission rates might be expected to increase. Furthermore, since no studies relating oxidant concentrations to hospital admission rates have been performed outside of Los Angeles, it is impossible at present to say whether oxidants influence admissions in other areas.

Aggravation of Existing Respiratory Diseases by Oxidant Pollution

AGGRAVATION OF ASTHMA

As a result of reports by physicians that asthmatic attacks are frequently associated with high-smog periods, Schoettlin and Landau[53] undertook a study to determine whether such a phenomenon indeed takes place. Five physicians selected 157 patients, 137 of whom participated in the study. All resided and worked in the Pasadena area. Fifty-four of the patients were younger than age 15. Most had had asthma for at least 5 years. Daily records of the time of onset and severity of asthma attacks were maintained by the patients between September 3 and December 9, 1956.

The Schoettlin and Landau study, as published, does not mention pollution measurement methods or averaging times used in data analysis. In *Air Quality Criteria for Photochemical Oxidants*,[39] it is assumed that oxidant levels in this study were daily instantaneous peak values as measured by the phenolphthalein method. Errata to the document were issued indicating that oxidant levels in this study were, in fact, determined by the KI method and not the phenolphthalein method.[16,40,47]

Since publication of *Air Quality Criteria for Photochemical Oxidants*[39] (AP-63) in 1970, a considerable effort has been made to determine with certainty the measurement methods and averaging times used in the Schoettlin and Landau study. Consultations with the authors have established that daily asthma attack rates were correlated with daily *maximum hourly average* oxidant levels, not with instantaneous peak levels. These consultations have also confirmed that oxidant measurements were indeed obtained from the Los Angeles County Air Pollution Control District, and from no other source. Careful examination of Air Pollution Control District records has further established that the District used only the potassium iodide method for measuring oxidant levels in Pasadena over the course of the Schoettlin and Landau study. Thus in the discussion to follow, Schoettlin and Landau are presumed to have used daily *maximum hourly average* oxidant concentrations, as measured by the *potassium iodide* method. In the discussion to follow, we also assumed that oxidant levels reported in this study correspond to daily maximum hourly levels and not to instantaneous peak levels, recognizing the error that might result if readings had corresponded to the latter.

In the Schoettlin and Landau study, the greatest number of asthma attacks occurred between midnight and 6 a.m., whereas the maximum oxidant levels were recorded between 10 a.m. and 4 p.m. Of the 3435 attacks reported, fewer than 5 percent were consciously associated with smog by the patients. No severe attacks were consciously associated with smog. One-third of the attacks consciously associated with smog were reported by a single patient. The correlation coefficient between daily attacks and the concurrent maximum oxidant reading was 0.37. The addition of other variables to the analysis did not significantly alter these results. Lagged correlations between oxidant concentration and the number of patients having attacks 6, 12, 18, and 24 hr later were generally lower than concurrent correlations. There was no significant difference between the average number of subjects having attacks on days above the median maximum hourly oxidant level of 250 $\mu g/m^3$ (0.13 ppm) ozone and the average number of patients having attacks on days when levels were below the median. However, the mean number of patients having attacks on days when daily maximum hourly oxidant levels exceeded 490 $\mu g/m^3$ (0.25 ppm) was significantly higher than on days when the daily maximum hourly oxidant level fell below this level. The authors suggested that this finding might indicate a threshold level for oxidants above which a physiologic response could be observed. Also, attack rates on days in which plant damage occurred were significantly higher than on days without plant damage. Specific oxidant levels associated with plant damage were not reported in this study, however. The effect of oxidant on asthma attack rates was most pronounced among persons who had lived in the area for 10 years or more. The data were examined further to see whether a small number of the subjects might be responsible for the observed correlation. The authors identified eight individuals (6 percent of the total panel) whose attacks corresponded most often to days on which plant damage occurred. Seven of these eight people were females. No other common characteristic could be discerned.

AGGRAVATION OF EMPHYSEMA AND CHRONIC BRONCHITIS

Several studies have been conducted to determine whether air pollution aggravates the condition of subjects suffering from chronic bronchitis and emphysema.

Motley et al.[38] reported a study of the lung function of 66 volunteers, 47 of whom had pulmonary emphysema. Before testing, subjects spent differing amounts of time in rooms from which oxidants had been removed by activated charcoal filters. Twenty-one subjects stayed in the filtered rooms for periods of 2 to 4 hr, 20 subjects for periods of 18 to 20 hr, and 25 subjects for periods of 40 to 90 hr. The study was performed over a 3½-year period in the late 1950's. Air quality measurements were provided by the Los Angeles County Air Pollution Control District. On the days of this study, maximum oxidant concentrations (presumably outdoors) between 9 a.m. and 2 p.m., calculated as ozone, ranged from 390 to 1040 μg/m³ (0.20 to 0.53 ppm) at monitoring stations several kilometers from the chamber. Maximum concentrations of other pollutants fell into the following ranges: Oxide of nitrogen, 0.2 to 0.6 ppm; sulfur dioxide, 0.05 to 0.25 ppm; and carbon monoxide, 5 to 27 ppm. Air was classified as smoggy when there was definite odor of ozone, reduced visibility, eye irritation, and the prediction of smog by the Los Angeles Air Pollution Control District. The investigators measured vital capacity, 3-sec forced expiratory volume (FEV 3.0), and maximal breathing capacity. Residual volume and air distribution measurements were also recorded.

An improvement in lung function, particularly a decrease in the residual lung volume, was observed in emphysematous subjects who had entered the filtered rooms on smoggy days and had remained in them for 40 or more hr. No significant changes in lung-volume measurements were observed when normal subjects breathed filtered air. No significant changes were observed when emphysematous subjects entered the chamber on nonsmoggy days. The smoking habits of subjects are not discussed in the report, but variations in these might have influenced the results, particularly if smokers were not allowed to smoke while in filtered rooms. The methods of analysis used in this study did not allow the effects of individual pollutants to be separated from one another.

At Los Angeles County Hospital, Remmers and Balchum[45] utilized a room with an air-conditioning system and a filter that could be used at the discretion of the investigators to remove photochemical oxidants, ozone, nitrogen oxides, and a portion of particulate matter from ambient air. Studies were conducted between July 1964 and February 1965. Analyses of total oxidant by the buffered KI method and of nitrogen dioxide by the Saltzman method were performed five times daily. Subjects performed lung-function tests one or more times daily while they lived in the room. In general, they spent 1 week in unfiltered air, a second week in filtered air, and a third week in unfiltered air. Throughout the study, air conditioning was adjusted to maintain a room temperature of 22° ± 1.4 °C (72° ± 2.5°F) and a relative humidity of 50 ± 5 percent.

Among other tests, determinations of airway resistance, carbon monoxide diffusing capacity, capillary oxygen tension, and oxygen consumption were performed while subjects were resting. Several of these determinations were repeated after subjects exercised.

Only four subjects were considered in the Remmers and Balchum report. All four had chronic lung disease. Two of them were considered to have relatively advanced emphysema but the other two were deemed to have only moderate pulmonary impairment. Smoking habits of subjects were not mentioned in the report of this study.

Beneficial effects of air filtration in studies of resting patients were most pronounced in the pair with only moderate pulmonary impairment. In this pair, airway resistance was about 20 percent lower during exposure to filtered air than to unfiltered air. A simultaneous increase in the speed of nitrogen washout also occurred in this pair. These changes were statistically significant at $\alpha = 0.10$.

The beneficial effects of air filtration in studies of exercising patients were slightly more pronounced in patients with advanced emphysema than in those with only moderate impairment. In all four patients, post-exercise oxygen consumption decreased significantly during exposure to filtered air. Capillary oxygen tension increased simultaneously in all four patients. Changes in oxygen tension were statistically significant only in patients with advanced emphysema. During studies of exercising patients, the mean oxidant concentration (calculated as ozone) was about 255 $\mu g/m^3$ (0.13 ppm) when air was not filtered, and about 40 $\mu g/m^3$ (0.02 ppm) when air was filtered.

For several reasons, the Remmers and Balchum study must be regarded as inconclusive. First, and probably most important, this report does not consider variations in the smoking habits of subjects. It has become quite clear that subjects were not allowed to smoke while breathing filtered air, but they were allowed to leave the study room and smoke, if desired, during their 2 weeks of exposure to ambient air.[43] Variation in smoking habits may thus have explained a major part of the physiologic difference observed between subjects exposed to filtered air and those exposed to ambient air.

Second, the ambient air to which subjects were exposed contained not only oxidants but other substances as well. Thus, even if the observed changes in lung function were attributable to air pollution, they might also be attributable, at least in part, to these other substances. Third, Remmers and Balchum appear to have analyzed data for only a very small number of subjects.

Though their report[45] considered only four patients, Remmers and Balchum collected data from 15 patients, most of them with relatively severe emphysema. Nine of these patients were smokers, five were non-smokers, and one stopped smoking when the study began. Patients generally spent about 1 week in unfiltered air, then a second week in filtered air, and finally a third week in unfiltered air. Analysis of the study of all 15 patients was performed for inclusion in the document, *Air Quality Criteria for Photochemical Oxidants.*[39] Results of that analysis are presented in Table 10-3, which shows coefficients of correlation between daily values of oxygen consumption and airway resistance (both measured under conditions of rest and exercise, with corresponding daily oxidant values measured in the morning and the afternoon). Each correlation coefficient was converted to a "t" statistic. The "t" statistics were then summed within pollution - test - specific categories. The investigators then derived a "z" statistic for each category by dividing the sum of "t" statistics by the sum's variance. The "z" statistics are presented in the last line of Table 10-3.

Using these methods, the authors observed a positive relationship, significant at $\alpha = 0.01$, between oxidant concentration and oxygen consumption during rest and exercise. They also observed a generally positive relationship, significant at $\alpha = 0.001$, between oxidant concentration and airway resistance measured at rest. As Table 10-3 shows, the relationship between airway resistance and afternoon oxidant concentrations was more consistently positive than that between resistance and morning concentrations.

In a published report of the study just described, Ury and Hexter[62] mention that each subject underwent a daily battery of 20 pulmonary function tests. The authors also mention that concentrations of three pollutants—oxidants, nitrogen dioxide, and nitric oxide—were measured. Though it is not explicitly stated, a distinct implication of the Ury and Hexter report is that oxidant levels were more strongly correlated with pulmonary function test results than were levels of either nitrogen oxide or nitric oxide. Of the 20 tests performed, only airway resistance is considered in this report. Thus the degree of correlation between pollution exposure and results of other tests cannot be determined, though the report implies that the other tests were not as strongly correlated with pollution exposure as was airway resistance. Finally, Ury and Hexter performed separate analyses for smokers and non-smokers. In smokers, the relationship between airway resistance and afternoon oxidant exposure, as tested by "z" statistic derived as described above, was significant at $\alpha = 0.002$. In nonsmokers, the relationship was significant at $\alpha = 0.001$.

In the Ury and Hexter report, unlike that of Remmers and Balchum, smokers are clearly separated from nonsmokers. That summed "z" statistics were more highly significant for nonsmokers tends to corroborate the conclusion of Remmers and Balchum, at least with respect to airway resistance. However, these findings must still be interpreted with some caution, since, as Ury and Hexter note, correlations between airway resistance and pollution exposure were significant at $\alpha = 0.05$ in only one-third of the individual subjects.

The relative effects of smoking and pollution on oxygen consumption are left largely unexplained.

The following average correlations between pollution exposure and oxygen consumption were computed from Table 10-3:

	Smokers	Non-smokers
Resting patients, a.m. oxidants	0.331	0.034
Resting patients, p.m. oxidants	0.231	0.175
Exercising patients, p.m. oxidants	0.292	0.055

That average correlations are consistently higher for smokers than for nonsmokers suggests that variations in smoking habits contributed at least partly to observed changes in oxygen consumption.

Rokaw and Massey[50] conducted a preliminary study of the effects of environmental variables on pulmonary function in a group of 31 patients in a chronic disease hospital in Los Angeles over a period of 18 months. A group of normal subjects was studied concurrently. All of the patients had chronic, nontuberculous respriatory diseases (predominantly pulmonary emphysema). Each subject underwent a series of pulmonary function tests four times weekly. In addition, function residual capacity by the helium method was determined monthly. Air pollution measurements were obtained from a station about 0.4 km (0.25 mile) upwind from the hospital. Concentrations of oxidant and oxidant precursor were measured by the KI method. Concentrations of ozone were also measured, but the method of measurement was not reported. Statistical methods were employed to detect associations between changes in pulmonary function and air pollution levels. The authors noted no consistent pattern of response to episodes of high pollution exposure, though in 6 of 31 patients, coefficients of correlation between daily pollution levels and corresponding pulmonary function were high enough to be termed "interesting." During the Rokaw and Massey study,[50] the mean odxidant concentration was 120 $\mu g/m^3$ (0.06 ppm), with a maximum of 820 $\mu g/m^3$ (0.42 ppm). These concentrations are quite moderate for the Los Angeles Basin and may well have been too low to promote any effect on lung function.

TABLE 10-3. CORRELATION OF MORNING AND EARLY AFTERNOON OXIDANT LEVELS WITH OXYGEN CONSUMPTION AND AIRWAY RESISTANCE OF 15 PATIENTS WITH CHRONIC RESPIRATORY DISEASE[45]

Patient's number and smoking history[a]	Number of observations		Maximum breathing capacity liters/min	Observed correlation coefficients				
				Oxygen consumption[b]			Airway resistance[c]	
				Resting		Exercising	Resting	
	Oxygen consumption	Airway resistance		a.m. oxidants	p.m. oxidants	p.m. oxidants	a.m. oxidants[d]	p.m. oxidants[e]
102 S	11	17		0.282	0.405	0.774	-0.379	-0.313
[103 NS]	14	14		0.123	0.210	0.251	0.717	0.567
[104 NS]	14	14		-0.313	0.007	0.258	0.638	0.641
106 S	17	17	88.4	0.473	0.579	0.521	-0.361	0.146
[107 S]	17	17	88.4	0.473	0.579	0.521	-0.361	0.146
[108 S]	18	18	99.4	0.489	0.448	0.409	-0.378	0.656
110 S	14	16	69.1	0.255	-0.136	-0.172	0.431	0.124
[111 NS]	15	17	117.7	0.434	0.092	-0.459	0.251	0.354
[112 S]	15	15	52.9	0.413	0.209	-0.348	0.339	0.433
[113 NS]	12	12	181.5	-0.107	0.222	0.088	0.034	0.006
[114 S]	13	13	65.3	-0.114	-0.158	-0.120	-0.161	0.058
[115 S]	14	14	26.3	0.423	0.290	0.130	0.217	0.557
[116 SS]	14	15	96.1	0.288	-0.094	0.751	0.715	0.460
[117 S]	15	15	38.6		0.189	0.456		0.609
[118 NS]	15	15	37.4		0.345	0.138		0.453
	"z"[f]			2.991[g]	2.761[g]	2.837[g]	3.621[h]	4.976[h]

[a] Smoking history S = smoker, NS = nonsmoker, SS = stopped smoking when study began. Brackets indicate patients who were tested during the same period.

[b] The first two correlations are with resting oxygen, which was measured around 11 a.m.; the third correlation is with exercise oxygen consumption, which was measured around 3 p.m.

[c] Airway resistance is measured while resting. Values given are averages of four measurements made throughout the day.

[d] a.m. oxidant was measured around 9:30 a.m.

[e] p.m. oxidant measured around 1:30 p.m.

[f] z-values were found by converting the individual correlations to t values, using the relationship $t_i = r_i[(n_i-2)/(1-r_i^2)]^{1/2}$ then summing the t values over all patients; the sums have variance $V(\Sigma t_i) = \Sigma[(n_i-2)/(n_i-4)]$. The z values shown are the ratio of $\Sigma(t_i)/[\Sigma(n_i-2)/(n_i-4)]$, which is approximately N(0,1).

[g] Significant at the 0.01 level.

[h] Significant at the 0.001 level.

The analyses presented in Rokaw's report are quite preliminary in nature. To the best of our knowledge, a thorough analysis of Rokaw's data has not yet appeared in the scientific literature.

Shoettlin[52] studied the effects of community air pollution, occupational exposure to air pollution, and smoking among Armed Forces veterans living in the Domiciliary Unit and Chronic Disease Annex of the Los Angeles Veterans Administration Center. Day-to-day variations in the physical status of men with chronic respiratory disease were studied in relation to changes in environmental conditions in the coastal area of the Los Angeles Basin. Two groups of men were selected for study. One of them consisted of 200 men with clinically determined chronic respiratory disease. The second group consisted of 200 asymptomatic men, matched on age and smoking history with the first group. Case-control pairs of men were studied weekly by means of repeated pulmonary function tests and responses to a respiratory symptom questionnaire. The study was performed between mid-August and mid-December 1958.

An air pollution monitoring station was set up at the site. Concentrations of total oxidant and oxidant precursor were measured by the KI method.

Multiple regression analysis showed no statistically significant effect of air pollution on respiratory function or symptoms. However, air pollutant measurements consistently explained more variation in symptom frequency and objective findings in the diseased group than in the control group. In this study, the statistical effect of maximal oxidant precursor on dependent variables was slightly stronger than the effects of other pollution variables measured.

Peters[44] examined pulmonary function in shipyard workers (welders, pipecoverers, and pipefitters) exposed to a variety of pollutants including metal fumes, asbestos, nitrogen oxides, and ozone. The mean concentration of ozone to which welders were exposed was 0.10 ppm (range 0.01 to 0.36 ppm); the mean concentration of nitrogen dioxide was 0.04 ppm (range 0.01 to 0.08 ppm). The results of the study supported the hypotheses (1) that chronic exposure to oxides of nitrogen and ozone among welders favors development of obstructive lung disease, perhaps in association with elastic recoil and increased residual volume, and (2) that chronic exposure to asbestos among pipecoverers promotes the development of restrictive lung disease (decreased total lung capacity). The pulmonary function of these groups was consistently less robust than that in pipefitters engaged in new ship construction who had minimal or no exposure to asbestos or welding fumes. These data suggest that chronic exposure to ozone or nitrogen oxides may contribute to the development of chronic lung disease. However, since welders were exposed to metal fumes and some asbestos as well as ozone and nitrogen oxides, the data do not allow a specific inference as to what the primary toxicant might have been.

DISCUSSION OF STUDIES OF AGGRAVATION OF EXISTING RESPIRATORY DISEASE

Available evidence tentatively suggests an association between exposure to ambient pollution in Los Angeles and both increased frequency of asthma attacks and decrements in pulmonary function among those with chronic lung disease. Studies published to date suggest that a maximum hourly oxidant concentration of 0.25 ppm may be sufficient to promote an increase in the proportion of asthamatics having attacks.

It must be noted clearly, however, that no study suggesting an association between oxidant exposure and exacerbations of chronic respiratory disease is without serious limitations. In studies performed to date, variations in the smoking habits of subjects have not been adequately considered. Also, investigators have tended to consider oxidant to the exclusion of other pollutants present in the air. It is possible that pollutants other than oxidant, acting singly or in combination with oxidant components, may have accounted at least partly for observed effects. Substantial further research in which covariates are appropriately treated and in which all pollutants present are considered must clearly be performed before the acute effects of photochemical oxidant pollution on persons with existing respiratory disease are fully understood.

Effects of Oxidants on the Promotion of Symptoms and Illness in Healthy Populations

SYMPTOM REPORTING AMONG STUDENT NURSES IN LOS ANGELES

Hammer et al.[18] examined symptom reporting in student nurses in relation to photochemical oxidant exposure. Freshman students at two nursing schools in Los Angeles were invited to participate in a prospective study of viral respiratory disease. To minimize bias, neither faculty nor students were informed that effects of air pollution were of major interest. Air quality was measured at monitoring stations located 1.5 to 3.0 km (0.9 to 2 miles) from both hospitals. Oxidant concentrations were measured by the KI method. Daily symptom diaries, which were collected each week, included questions related to headache, eye discomfort, cough, and chest discomfort. Symptoms were graded mild, moderate, or severe. Symptom reporting covered the period October 1961 through June 1964. A daily average of 61 students participated.

Patterns of symptom reporting from both schools were similar and were pooled in all analyses. All three grades of symptoms were considered positive reponses, although the occurrence of moderate or severe symptoms was rare. Simultaneous daily measurements of oxidants, carbon monoxide, nitric oxide, nitrogen dioxide, sulfur dioxide, and maximum daily temperature were available on more than 90 percent of the 868 days in this study.

Results of the study are presented in Table 10-4. Simple headache frequency rose slightly at and above oxidant concentrations of 0.25-0.29 ppm. The frequency of simple eye discomfort, however, increased as daily maximum hourly photochemical oxidant levels exceeded 294 to 372 $\mu g/m^3$ (0.15 to 0.19 ppm). Simple rates of cough remained relatively constant until the maximum hourly oxidant reached 588 to 764 $\mu g/m^3$ (0.30 to 0.39 ppm), at which level the rate began to increase fairly steeply. The simple rate of chest discomfort began to rise at an oxidant concentration of about 0.25 to 0.29 ppm.

The authors also computed adjusted symptom rates by excluding days in which student nurses reported a fever. For all symptoms except headache, the relationship of adjusted symptom rates to oxidant exposures was similar to that of simple rates. Temperature-fever adjusted headache rates increased slightly at daily maximum hourly oxidant levels of 0.15-0.19 ppm and increased more steeply as oxidant levels reached 588 to 764 $\mu g/m^3$ (0.30 to 0.39 ppm).

Symptom frequencies in this study were more closely related to photochemical oxidants than to carbon monoxide, nitrogen dioxide, or daily minimum temperature. Oxidant concentrations at which rates of eye discomfort were observed to increase were comparable to those observed in other published studies. Oxidant concentrations at which cough and chest discomfort rates were observed to increase were quite similar to ozone concentrations observed to produce impairment of pulmonary function and respiratory irritation in Bates' experimental studies of humans.[1] In these experimental studies, healthy males performing intermittent light exercise were exposed to 725 $\mu g/m^3$ (0.37 ppm) ozone for 2 hr. The tolerance to the effects of photochemical oxidants of healthy young adults like the student nurses studied by Hammer et al. may be different from that of other population segments like the aged, the very young, the ill, and the pregnant.

The results of Hammer's study[18] arouse interest as to what the effects of photochemical oxidants on these other population segments might be.

TABLE 10-4. RELATIONSHIP OF AVERAGE DAILY SIMPLE AND ADJUSTED[a] PERCENTAGE OF STUDENT NURSES REPORT TO PHOTOCHEMICAL OXIDANT LEVELS FOR 868 DAYS, NOVEMBER 1961 THROUGH MAY 1964[18]

Daily maximum hourly oxidant level, ppm[b]	Number of days	Average number of nurses reporting daily	Average daily percent of symptoms reported							
			Headache		Eye discomfort		Cough		Chest discomfort	
			Simple	Adjusted	Simple	Adjusted	Simple	Adjusted	Simple	Adjusted
≤ 0.04	229	64	16.5	10.5	8.6	5.0	12.7	9.1	3.5	1.8
0.05 - 0.08	184	59	16.3	10.7	9.2	5.4	13.4	9.9	3.6	1.8
0.09	35	58	16.0	10.6	9.0	5.6	13.3	10.2	3.2	1.9
0.10 - 0.14	176	62	15.6	11.0	8.6	5.9	11.9	9.4	3.4	1.8
0.15 - 0.19	144	58	15.7	11.4	10.0	6.9	12.3	9.7	3.7	1.7
0.20 - 0.24	63	60	15.6	11.6	12.2	9.2	11.6	9.1	3.1	1.6
0.25 - 0.29	25	60	16.7	11.5	14.9	11.2	12.4	9.6	3.6	2.0
0.30 - 0.39	9	67	16.9	13.4	21.1	17.8	15.2	11.7	4.1	2.3
0.40 - 0.50	3	53	16.8	15.0	35.0	31.8	19.3	16.9	7.0	5.8
Overall average		61	16.1	10.9	9.6	6.3	12.6	9.5	3.5	1.8

[a] All days on which the system was reported along with "feverish," "chilly," or "temperature" are excluded.

[b] 1 ppm = 1960 $\mu g/m^3$.

EFFECTS OF OXIDANTS ON NEW ILLNESS IN COLLEGE STUDENTS

Durham[12] reported a study of the short-term effects of air pollution on the health of students at seven California universities—five in the Los Angeles area, and two in the San Francisco Bay Area. The Los Angeles area generally experiences higher pollution concentrations than the Bay Area. All seven universities participating in Durham's study were located within 8 km (5 miles) of a weather station and a pollution monitoring station. Nine indices of weather and eight indices of pollution were considered. The method by which oxidant concentrations were measured was not reported, although presumably the method was KI, at least in the Los Angeles area.

Health data were collected during the 1970-71 school year. Each time a student visited the student health service with a health-related complaint a sheet requesting demographic information, smoking habits, and physician's diagnosis was completed. Diagnoses were assigned to 1 of 14 categories, including eye irritation, respiratory and allergic diagnoses, and gastroenteritis (a diagnosis not expected to be related to pollution exposure). Only first visits with individual illnesses were included in data analysis. About 22 percent of collected data sheets showed no physician's diagnoses. In all, 11,659 sheets with first-visit respiratory diagnoses were received.

For each school, the author computed coefficients of correlation between levels of pollution and weather variables on 1 day and proportions of new illness on the same day and on days up through 7 days later.

In general, correlations of pollution with bronchitis were greatest when lagged 5 or 6 days; correlations of pollution with combined respiratory disease were greatest when lagged zero to 3 days; and correlations with asthma, eye irritation, headache, and hay fever were greatest when lagged zero days or 1 day.

Between 20 and 25 percent of pollution/respiratory illness correlations were statistically significant at $\alpha = 0.05$, whereas only 5 percent of pollution/gastroenteritis correlations were significant. The illnesses most strongly associated with pollution were, in descending order, pharyngitis, bronchitis, tonsillitis, colds, and sore throat. The pollutant variables most strongly associated with illness in general were, in descending order, peak oxidant, mean sulfur dioxide, mean nitrogen dioxide, and mean nitric oxide. Stronger associations between pollution and illness were generally observed in the Los Angeles area than in the Bay Area. The authors attributed this finding to the difference in pollution exposure between the two areas, although pollution measurements in the two areas were not compared in the report.

In a separate analysis, the days of the study were separated into high- and low-pollution days for each school in the Los Angeles area. High-pollution days were defined as days on which concentrations of at least half the measured pollutants exceeded their means by at least 0.25 standard deviation. Days on which concentrations of at least half the measured pollutants were less than 0.25 standard deviation below their means were assigned to the low-pollution category. For each school, the authors calculated ratios of upper respiratory illness rates occurring on or following high-pollution days to illness rates occurring on or following low-pollution days. At the University of California, Irvine, which consistently experienced lower pollution levels than any other school in the Los Angeles area, this ratio was very close to 1.0 for all lag periods from zero through 7 days between the occurrence of pollution and the onset of symptoms. At the University of Southern California (USC), which experienced the highest pollution levels of any school, this ratio was larger than that observed at Irvine for all lag periods except zero days. On the average, the ratio at USC exceeded the ratio at Irvine by 16.7 percent. The largest interschool difference in ratios, about 50 percent, occurred at a lag period of 6 days. The methods of analysis used in this study did not permit inferences as to the lowest oxidant levels necessary to promote increased illness rates.

OXIDANTS AND ELEMENTARY SCHOOL ABSENTEEISM

Wayne and Wehrle[64] examined the effects of oxidant air pollution on respiratory illness among children as measured by absentee rates from elementary school. Absentee data were collected from two elementary schools in Los Angeles throughout the 1962-63 school year. Reasons for absences were intially obtained from parents and were classified into various categories by teachers. Air pollution measurements were supplied by the Los Angeles Air Pollution Control District. Oxidant concentrations were measured by the KI method. Measurements for school 1, located in east downtown Los Angeles, were made at a station located within 3 km (2 miles) of that school. Air quality data for school 2 were monitored at a station within 3 km (2 miles) of the school during the initial 3 months of study and at a station 6.5 km (4.5 miles) from the school for the remainder of the study. During this study, daily oxidant concentration averaged from 10 a.m. to 3 p.m. ranged from nearly zero to about 0.23 ppm. This concentration exceeded 0.20 ppm on 6 (1.8 percent) of a possible 336 days. Oxidant was the only pollutant considered in this study.

Absence rates in both schools were highest during the winter season, when levels of oxidants were generally lowest. No consistent association was observed between weekly mean oxidant levels and weekly absence rates for respiratory disease. The highest absence rates were reported on Mondays and Fridays at both schools; in contrast, oxidant levels were consistently higher during midweek periods. In a further analysis, correlations were computed between uncorrected daily absence rates and same-day average oxidant concentrations between 10 a.m. and 3 p.m. In a second analysis, absence rates corrected for day-of-week effect were correlated with same-day 10 a.m. to 3 p.m. oxidant concentrations and with corresponding concentrations occurring 1 day before. Results from the two schools were kept separate in all analyses. No analysis revealed a positive association between oxidant concentration and absence rates.

EFFECT OF PHOTOCHEMICAL OXIDANTS ON SYMPTOM RATES IN JAPANESE ELEMENTARY SCHOOLCHILDREN

Shimizu[55] discussed the effects of photochemical smog exposures on symptom reporting rates at two junior high schools in Osaka Japan, which will be designated schools A and B. At school A, an unusually high number of students complained of symptoms on 3 days in the fall of 1972 (September 13, 21, and 22). An unusually high number of eighth graders at school B complained of symptoms on October 3, 1972. At each school, symptom questionnaires were distributed on the day or the day after the unusual complaint rates occurred. The questionnaires inquired about the presence of nine symptoms, including eye irritation, respiratory discomfort, cough, dizziness, nausea, and numbness of the extremities.

At school A, questionnaires appear to have been distributed to all students. However, they were collected only from students who reported the presence of at least one symptom. Faced with uncertainty as to the appropriate denominator, the author selected the total enrollment of school A, (1313 students) as the basis for calculation of symptom rates.

Maximum concentrations of pollutants measured at a station near school A (Station P) on September 13 were as follows:

Pollutant:	*ppm*
Oxidant (3 p.m.)	0.14
Nitric oxide (2 p.m.)	0.17
Nitrogen dioxide (2 p.m.)	0.12
Sulfur oxide (1 p.m.)	0.6

The maximum temperature, 29.4°C (85°F), was recorded at 11 a.m. At another nearby station (Station O), a maximum oxidant concentration of 0.19 ppm was recorded between 2 and 3 p.m. Only Station O, which measured only oxidant, was operating on September 21 and 22. At this station, the maximum oxidant concentration on September 21 was 0.17 ppm, recorded at 1 p.m. On September 22, the maximum oxidant concentration was 0.11 ppm, recorded at 12 noon. Aerometric measurement methods were not reported.

At school A, a total of 263 students (20.0 percent) reported at least one symptom on September 13. Corresponding totals on September 21 and 22 were, respectively, 133 (10.1 percent) and 72 (5.5 percent).

It is of interest that on September 13, the rate of symptom reporting in physical education classes increased as concentrations of oxidants, NO, and NO_2 increased. Physical education classes were conducted outdoors. The average rate of symptom reporting among students not having physical education classes that day and students having class in the morning was 7.5 percent. The highest rate of symptom reporting, 50.9 percent, was observed in the class which met between 1 and 2 p.m. Peak levels of NO and NO_2 were recorded during this hour. As mentioned, the peak level of sulfur oxide had been recorded at 1 p.m. In the class that met 1 hr later, when peak levels of oxidant were recorded, the rate of symptom reporting dropped slightly to 45 percent. On September 21 and 22, when both oxidant concentrations and symptom rates were somewhat lower than on September 13, symptom rates were distributed randomly among physical education classes. Unfortunately, concentrations of pollutants other than oxidant were not measured on these two days.

The author noted that on September 13, not only the rate of symptom reporting but also the distribution of symptoms changed with physical education class. Among students not having physical education class or having it in the morning, 94.6 percent of students with symptoms reported eye irritation. Only 37.5 percent of these students complained of sore throat, and 19.6 percent complained of coughing. In the physical education class meeting between 1 and 2 p.m., however, only 50 percent of students with symptoms reported eye irritation, while 81.0 percent reported sore throat, 60.7 percent reported coughing, and 59.5 percent reported chest discomfort. These findings suggested to the author that the components of photochemical pollution that promote eye irritation may be different from those that promote respiratory symptoms.

At school A, the majority of students with symptoms on September 21 and 22 complained of eye irritation.

At school B, as mentioned, an unusually high number of eighth-grade students complained of symptoms on October 3, 1972. At two nearby stations, maximum oxidant concentrations of 0.09 and 0.07 ppm were recorded on that day. No mention of other pollutant concentrations was made in Shimizu's report. Of 248 students surveyed, 152 (61.3 percent) reported some type of symptom. The pattern of symptom reporting at this school was somewhat different from either pattern observed at school A. At school B, 53.5 percent of males and 71.7 percent of the females surveyed reported symptoms, whereas at school A there had been little difference in symptom rates between the sexes. In school B, higher percentages of students with symptoms than in school A reported such nervous-system-related symptoms as headache, dizziness, nausea, and numbness of the extremities (which can result from hyperventilation). These symptoms, like chest discomfort occurred more frequently in females than in males. In school B, only 21.7 percent of students with symptoms reported eye irritation.

Shimizu's findings arouse interest as to whether the components of photochemical air pollution that promote nervous-system-related symptoms like those described above are different from either the components that promote eye irritation or those that promote respiratory symptoms. As yet, however, these findings are far from conclusive, particularly since pollutants other than oxidants were in the ambient air during this study.

Makino and Mizoguchi[32] reported a study, performed over the year July 1972 to June 1973, of the influence of air pollution exposures on rates of symptom reporting in students. This study included 854 students, of whom 110 were in kindergarten, 327 were in elementary school, 335 were in junior high school, and 82 were in senior high school. All students attended schools in the northern part of Tokyo. Each day they completed a questionnaire inquiring about the presence of 17 symptoms, including eye irritation, irritation of the upper respiratory tract, cough, phlegm production, fatigue, headache, and fever. Daily rates of symptom reporting were correlated with same-day pollution concentrations. We believe that the pollution concentrations used in statistical analyses were daily maximum hourly concentrations, but this is not clearly stated in the report. Measured pollutants included oxidant, nitric oxide, nitrogen dioxide, sulfur dioxide, and micro-particulates. Measurement methods were not reported. Daily maximum hourly daytime temperature and minimum hourly daytime humidity were also considered.

The data were analyzed in several different ways. In the first analysis, rates of symptom reporting on two days of high pollution, one in July and one in August, were computed. On the first day, maximum hourly concentrations of oxidant, nitrogen dioxide, nitric oxide, and sulfur dioxide were, respectively, as follows: 0.17, 0.10, 0.10, and 0.03 ppm. On the second day, maximum hourly concentrations of O_x, NO_2, NO, and SO_2 were, respectively, as follows: 0.21, 0.07, 0.09, and 0.14 ppm. On both days, rates of nearly all symptoms were higher than corresponding monthly average rates. The elevation in rates was most marked for eye irritation, which an average of 10.7 percent of students reported on the two high-pollution days, versus an average of 1.6 percent in the months of July and August. Rates of sore throats (average of 4.0 percent on high-pollution days versus 0.65 percent in corresponding months) and headache (2.9 vs. 1.0 percent) were also elevated noticeably.

In the next analysis, individual environmental measurements were correlated with same-day symptom rates within each month of the study. Of 105 oxidant/symptom correlations, 37 (35 percent) were significant at $\alpha = 0.05$. Of these, 14 (13 percent) were significant at $\alpha = 0.001$. Of 98 SO_2 symptom correlations, 22 (22 percent) were significant at $\alpha = 0.05$, and 10 (10 percent) were significant at 0.001. Of 105 NO_2/symptom correlations, 22 (21 percent) were significant at $\alpha = 0.05$, and none were significant at $\alpha = 0.01$. Of 116 temperature/symptom correlations, 19 (16 percent) were significant at $\alpha = 0.05$. Only 5 (6 percent) of 88 NO/symptom correlations were significant at $\alpha = 0.05$. The directions of these correlations were not presented.

When these types of correlations were computed over the whole year of study, 6 (46 percent) of 13 oxidant/symptom correlations were significant at $\alpha = 0.01$. All coefficients were positive. The association between oxidant exposure and symptom rate was most marked for dyspnea (for which the oxidant/symptom correlation was 0.653) and for eye irritation, lacrimation, and sore throat. For each of these latter three symptoms, the oxidant/symptom correlation was 0.621 ($p < 0.001$). No yearly symptom/pollution correlations were significant at $\alpha = 0.05$ for SO_2, NO_2, or NO. Seven of 13 (54 percent) yearly temperature/symptom correlations were significant at $\alpha=0.05$. All seven were positive. Coefficients of correlation between oxidant concentrations and symptoms were generally larger than those between temperature and symptoms.

In another analysis, pairs of environmental variables were correlated with same-day rates of symptom reporting. Correlations between oxidant/NO_2 and symptom rates were significant at $\alpha = 0.05$ in 9 of 13 cases (69 percent). Correlations between oxidant/SO_2 and symptom rates were significant in 7 of 13 cases (54 percent), as were correlations between oxidant/temperature and symptom rates. Environmental variable pairs that did not contain oxidant were significantly correlated with symptoms less often and less strongly than those that contained oxidant.

In another analysis, the authors compared symptom rates on days when the maximum hourly oxidant level exceeded 0.10 ppm to rates on days when it did not. Such comparisons are shown for six symptoms in Figure 10-4. As the figure shows, symptom rates, particularly rates of eye irritation and sore throat, were quite consistently elevated on the higher-oxidant days. The report does not clearly state why the months of January through April 1973 are missing from the line showing symptom rates on higher-oxidant days in Figure 10-4. It is probably safe to surmise, however, that since these are winter or early spring months, they contained very few days on which the oxidant concentrations exceeded 0.10 ppm.

Mizoguchi et al.[37] investigated the effects of short-term air pollution exposure on 515 students in a junior high school in southeastern Tokyo. Over a 2-month period between May and July 1974, these students completed a daily questionnaire inquiring about the presence of 17 symptoms of the respiratory and other systems. Air pollution measurements were made on the school grounds. Oxidants were measured each hour by the KI method; ozone was measured by (presumably ethylene) chemiluminescence; and nitric oxide and nitrogen dioxide were measured by the Saltzman method. Concentrations of eight other pollutants were also measured. The maximum hourly oxidant level observed during this study was 0.23 ppm. No frequency distribution of pollutant concentrations was presented.

In still another analysis, Makino and Mizoguchi compared symptom rates on days when the oxidant concentration exceeded 0.15 ppm to rates on days when oxidant concentration did not achieve 0.10 ppm. Results of this comparison are shown in Figure 10-5. As expected from the results of other analyses of these data, increases in symptom rates on the higher-oxidant days were most marked for eye irritation, sore throat, headache, and coughing.

Three types of statistical analyses were performed on data gathered from all students. In the first type, a simple correlation analysis, daily maximum hourly measurements of each pollutant, were correlated with the proportion of students reporting each symptom on the same day the pollution measurements were made. Correlation coefficients statistically significant at $\alpha = 0.001$ ($r \geq 0.5$) were computed between oxidant concentrations and 5 of 17 symptoms, including eye irritation, shortness of breath, sore throat, headache, and blurred vision. Positive coefficients significant at $\alpha = 0.01$ ($r \geq 0.35$) were computed between oxidant concentrations and seven other symptoms. Ozone was found to correlate significantly at $\alpha = 0.001$ with three symptoms (eye irritation, shortness of breath, and sore throat) and at $\alpha = 0.01$ with seven other symptoms. Sulfur dioxide, like oxidant, was found to correlate significantly with 12 of 17 symptoms at the 0.01 level. Suspended particulate matter correlated significantly with 13 symptoms. Daily minimum relative humidity correlated negatively and significantly with 15 symptoms. No correlations of pollutants with other pollutants and with meteorologic variables were presented.

In the second analysis, daily proportions of each individual symptom were correlated multiply with pairs of same-day measurements of environmental variables. (The report was unclear as to whether all possible environmental-variable pairs or only selected pairs were considered.) Six such pairs were correlated significantly at $\alpha = 0.001$ ($r \geq 0.6$) with both eye irritation and shortness of breath. Oxidant was a member of all 12 of these pairs. Seven pairs, of which six contained oxidant, were correlated significantly with sore throat. Five pairs correlated significantly with hoarsness, but only one of them contained oxidant. None of the pairs of environmental variables that correlated significantly with symptom rates contained ozone, but whether ozone was included in any of the tested pairs is not clear.

The third analysis was a principal components analysis. Three environmental principal components were derived. The first was loaded heavily for oxidant, ozone, and sulfur dioxide; the second was loaded heavily for most pollutants except oxidant and ozone (the second also included

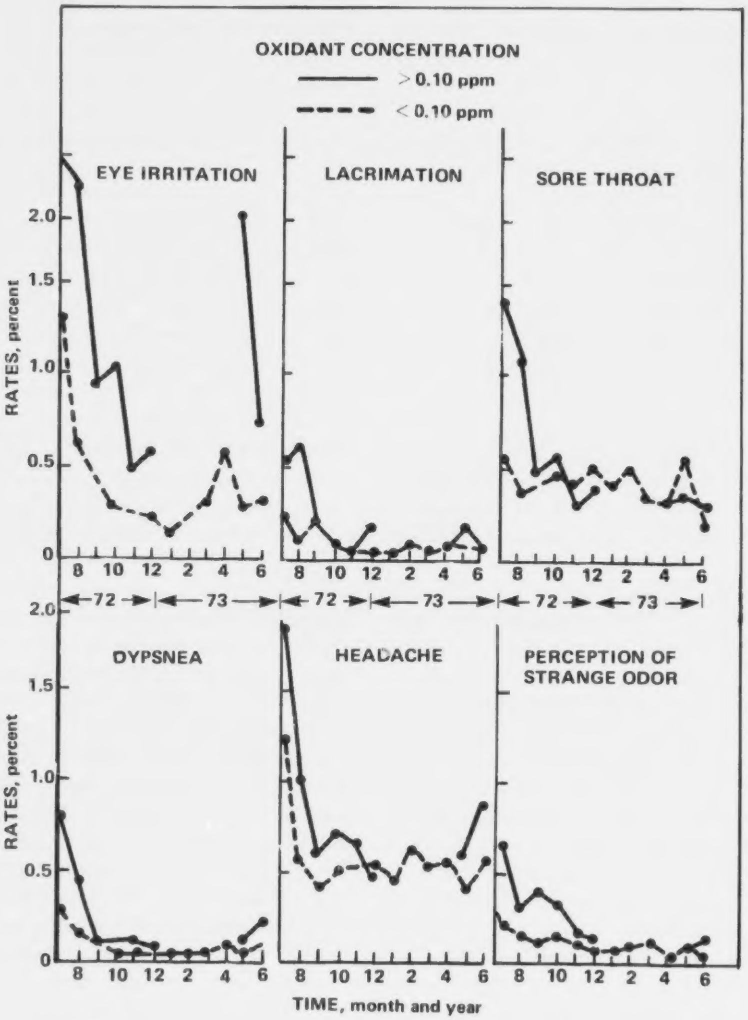


Figure 10-4. Monthly changes in symptom and complaint rates with daily maximum hourly oxidant content above and below 0.10 ppm.[32]

sulfur dioxide); and the third was loaded heavily for temperature and carbon monoxide. Nearly all symptoms, particularly such irritation symptoms as eye irritation, sore throat, hoarsness, cough, and phlegm, were associated strongly with the first principal component. No symptoms were strongly associated with either of the other two components.

From the overall study sample, the authors selected a group of 74 students with allergic tendencies and a group of 47 with orthostatic dysregulation (O.D.). In the report, the author stated that O.D. is not the same entity as orthostatic hypotension, though he did not define the condition further. O.D. appears to be a condition of unusually intense reactivity to stress. Over the entire study period, the group with O.D. reported noticeably but not significantly higher rates of cough, headache, and sneezing than either of the allergic group or the students as a whole. Symptom rates in the allergic group were generally higher than in the students as a whole, but not as markedly as in the group with O.D. On a day when maximum hourly oxidant achieved a concentration of 0.23 ppm, rates of symptom reporting increased in all groups. Increases in symptom rates were largest in the O.D. group, particularly for eye irritation, cough, and headache. Again, symptom rates in the allergic group were intermediate between the students as a whole and the group with O.D. In this report, the author mentions (though does not reference) a concurrent investigation in which no effect of air pollution exposure on pulmonary function was observed. This finding is consistent with the hypothesis that subjective symptoms may sometimes reflect pollution exposures more accurately than objective parameters.

The Japanese Environment Agency conducted a large survey of student health in 1975. Results were drawn from schools in seven prefectures throughout the country. A preliminary analysis of these results has been reported.[21] The period covered by this survey is not clearly stated, but it

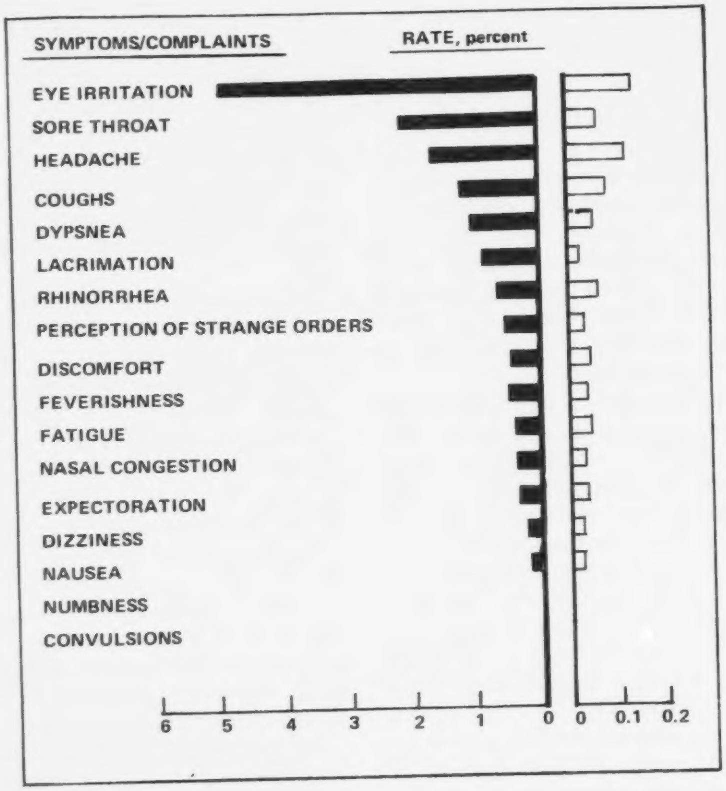


Figure 10-5. Comparison of various symptom and complaint rates on days when oxidant concentrations were over 0.15 ppm and below 0.10 ppm.[32]

appears to have been about 180 days. The majority of students included in the survey were in junior high school, although students in elementary and senior high school were also included. Unfortunately, the report of the survey often presents numbers of people reporting illness, but not rates of illness. Rates cannot be computed since the report does not present the total number of persons included in the survey. Also, the report does not present the methods used to generate aerometric data, so its findings are quite difficult to compare with findings made in the United States.

The primary survey tool was a symptom questionnaire (in some prefectures self-administered, and in others interviewer-administered) that was completed by students who complained of symptoms at school. It must be remembered that this survey was based primarily on students who voluntarily reported symptoms. No systematic effort was made to determine either the extent of nonresponse or the effects that nonresponse bias may have exerted on the results. Thus, as the authors acknowledge, the majority of findings of this survey, though interesting and suggestive, must be considered preliminary and inconclusive.

The most common symptom reported was eye irritation which, on the average, constituted about 36 percent of all symptoms reported. About 30 percent of all symptoms reported were general symptoms, including numbness in the extremities, headache, dizziness, nausea, and lethargy. Irritation of the nose and throat constituted about 20 percent of all reported symptoms. Respiratory symptoms, including cough, chest pain, and phlegm production, also accounted for about 20 percent of all reported symptoms.

In Saitama prefecture, 10 schools containing 7440 students were included in the survey. On days when the oxidant concentration never exceeded 0.15 ppm, an average of only 0.052 percent of students reported smog-related symptoms. On days with oxidant concentrations of at least 0.15 ppm but less than 0.25 ppm, this percentage rose to 0.48 percent. On two days when oxidant concentrations were at least 0.25 ppm, an average of 16.4 percent of students reported symptoms. A similar (though less strictly designed) survey conducted in Kanagawa prefecture apparently yielded similar results. In one elementary school, 140 of 1510 students (9 percent) complained of smog-related symptoms on one afternoon when the following pollution measurements were made at a nearby monitoring station: oxidant, 0.253 ppm; nitrogen dioxide, 0.018 ppm; and oxides of sulfur, 0.042 ppm. (This is the only instance in the report in which measurements of pollutants other than oxidants are presented.) Eleven students reporting feelings of choking and numbness in the extremities were taken by ambulance to a nearby hospital. Detailed family and personal histories were obtained from five hospitalized students. Of these, all had positive personal histories of allergic conditions, and four had positive family histories.

DISCUSSION OF OXIDANT EFFECTS ON PROMOTION OF SYMPTOMS AND ILLNESS IN HEALTHY POPULATIONS

A consistent positive association between oxidant concentrations and rates of new illness and symptoms has been observed both in the United States and in Japan. The Hammer et al. study of student nurses[18] suggests that rates of chest discomfort and cough begin to increase at maximum hourly oxidant concentrations of 0.25 to 0.29 ppm and 0.30 to 0.39 ppm, respectively. As mentioned, this finding is quite consistent with the results of the human experimental studies by Bates et al., in which decrements in pulmonary function and respiratory irritation have occurred at an ozone level of 0.37 ppm. Recent Japanese studies suggest that cough, chest discomfort, and other symptoms may occur in school children at oxidant concentrations between 0.10 and 0.15 ppm.

The consistency between Hammer's epidemiologic observations and Bates[1] experimental findings suggests that ozone alone may well account for some of the symptoms associated with oxidant exposures in epidemiologic studies. However, it appears quite clear that ozone does not account for all the symptoms observed in the field. (Indeed, that ozone does not produce the eye irritation so often associated with photochemical oxidants has been quite well established.) Results of the Japanese studies cited above (in which correlations between total oxidant and symptom reporting rates were larger and more consistently significant than ozone/symptom correlations) bring nonozone components of photochemical pollution under suspicion.

As mentioned, Shimizu[55] observed that the majority of students reporting symptoms in the morning of a high-pollution day complained of eye irritation. By the afternoon of the same day, when ozone concentrations had risen above morning levels, the majority of students with symptoms complained of respiratory discomfort. This observation, coupled with the findings of Bates and Hammer, appears to favor the hypothesis that certain oxidants other than ozone, though perhaps including ozone precursors, produced the eye irritation experienced in the morning, and that ozone was an important contributor to the respiratory irritation experienced in the afternoon.

On the basis of findings presented here, it appears that lower levels of oxidant pollution affect Japanese subjects more than American subjects. One or both of the following factors may explain this disparity. First, the components of Japanese oxidant pollution may be different from corresponding components in the United States. For example, high sulfur oxide levels appear to accompany high oxidant levels more frequently in Japan than in the United States (e.g., Los Angeles). Second, occurrences of elevated pollution appear to receive considerably more publicity in Japan than United States. This publicity may influence symptom reporting. For these reasons, the results of Japanese and American studies should be compared with caution, particularly in view of the apparent discrepancy between the Japanese studies and the Wayne and Wehrle study of absenteeism.[64]

The Durham study[12] and the Japanese studies cited above arouse interest as to whether photochemical oxidant pollution predisposes young populations to the development of increased rates of chronic respiratory disease in adulthood. Unfortunately, the relationship between acute illness or symptoms in childhood and chronic respiratory illness in adulthood is not at all clearly characterized as yet. Until it has been characterized, the question as to whether transient irritative symptoms constitute a bona fide impairment of health must remain open.

Impairment of Performance Associated with Oxidant Pollution

ATHLETIC PERFORMANCE

Wayne et al.[65] have studied the athletic performance of student cross-country track runners in 21 competitive meets at a high school in Los Angeles County from 1959 to 1964. Aerometric data were supplied by the Los Angeles County Air Pollution Control District. Oxidant measurements for the hour of the race, and for 1, 2, and 3 hr before the race were related to the percentage of athletes who did not improve their running times from the previous meet. Oxides of nitrogen, carbon monoxide, particulates, temperature, relative humidity, wind velocity, and wind direction were also considered, but they bore less relationship to performance than did oxidant. Evidently, correlations between performance and sulfur dioxide were not computed.

The authors observed a significant positive relationship between hourly oxidant levels and the percentage of team members whose performance failed to improve from the previous home meet. This relationship is shown in Figure 10-6. As the figure shows, the proportion of runners failing to improve their times rose as concentrations of oxidant in the hour before the race increased over a range of 60 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm). However, convincing linear relationships between oxidant concentration and performance could not be discerned within individual portions of this concentration range. For both 1959-61 and 1962-64, the coefficient of correlations between oxidant concentration and percentage of runners failing to improve their times was 0.945. As the interval between the time of oxidant measurement and the race increased, corresponding correlation coefficients decreased.

The results of this study strongly suggest an effect of some component of the pollution measured as oxidant on team performance. Speculating as to possible mechanisms underlying the observed association, Wayne et al. cited the possibility that some component of photochemical smog might elevate the body's demand for oxygen during exercise. They also felt that breathing photochemical air pollutants might produce increased airway resistance or might lead to discomfort, which might in turn limit the runners' motivation.

In an analysis performed at the University of North Carolina, Herman[23] also investigated the effects of air pollution on performance of cross-country runners. In his analysis, Herman used the same data that Wayne et al.[65] had used from the cross-country seasons of 1959-64 as well as data from the seasons of 1966-68. The dependent variable in Herman's analysis was the average speed attained in each meet by each runner who participated in all home meets in a given season, corrected both for the runner's average speed over the season and for the teams'average speed over the season. This differed from the dependent variable used by Wayne, which was the percentage of runners who had failed to improve their times from the previous meet.

Herman constructed regression models that incorporated all data from 1959-68 and that employed the following as independent variables: hourly air pollution measurements, the individual running seasons, the number of days since the first home meet of the season, the square of this number of days, the maximum daily temperature, and the square of this temperature. Stepwise regressions in which pollution measurements were entered into the model both before and after temperature variables were performed. In both cases, an inverse association between running speed and oxidant measured in the hour before the meet (significant at α = 0.0001) was observed. Little association between running speed and total suspended particulates, carbon monoxide, and oxides of nitrogen was observed.

Folinsbee and his associates[14,15] have conducted two experimental studies that suggest a mechanism underlying Wayne and Wehrle's epidemiologic observations. These studies are described in detail in the preceding chapter. In the first study, healthy young adults (20 males and 8 females, 18 nonsmokers and 10 smokers) were assigned to one of six exposure groups. Two groups of healthy young adults received a 2 hr exposure to 0.37 ppm ozone; two other groups were exposed to 0.50 ppm; and two others were exposed to 0.75 ppm. At each exposure level, one group rested throughout exposure, and the other underwent alternating 15-min periods of rest and exercise. Each of the six groups was also subjected to a mock exposure while adhering to its experimental protocol during actual exposure. At the end of all exposures and mock exposures, all subjects underwent a submaximal exercise test.

Respiratory frequency significantly increased over control values after intermittent exercise at all exposure levels and with resting exposures to 0.75 ppm ozone. Tidal volume decreased significantly after intermittent exercise at the 0.50- and 0.75-ppm exposure levels. Forced vital capacity decreased significantly after intermittent exercise at 0.50 ppm ozone, and after both rest and exercise at 0.75 ppm. Maximum expiratory flow rate at 50 percent of vital capacity decreased significantly after intermittent exercise at all three exposure levels. Many subjects reported common symptoms of ozone exposure, including sore throat and

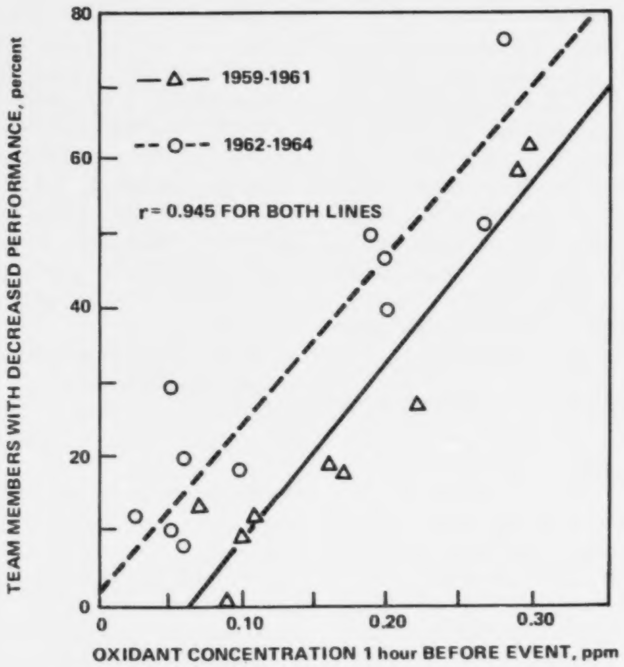


Figure 10-6. Relationship between oxidant concentration in the hour before an athletic event and percentage of the team failing to improve running time.[65]

cough. Symptom severity increased with exposure level and exercise. No significant changes in minute volume or in oxygen uptake were observed after the submaximal exercise tests employed in this study.

The authors believed that because of the speed with which they developed, the observed physiologic changes were due to reflex bronchoconstriction mediated by irritant receptors and perhaps by Hering-Breuer (stretch) receptors in the lung.

In the second study of Folinsbee et al.,[14] healthy young adult males were exposed to 0.75 ppm ozone for 2 hr. During exposure, subjects alternately rested for 15 min and lightly exercised for 15 min. In random order, each subject also underwent a 2-hr control exposure to filtered air. At the conclusion of all exposures, subjects exercised until exhausted.

Values of the following parameters, measured during maximal exercise, were significantly lower after ozone exposure than after filtered air exposure: maximum work load attained, heart rate, minute volume, tidal volume, and oxygen uptake.

The authors believed that the most likely mechanism underlying the results observed was that ozone produced stimulation of irritant receptors in the lung, which in turn produces restriction of inspiratory reserve volume, reduction of maximal tidal volume, and a modest increase in airway resistance.

In the light of Folinsbee's results, it is quite conceivable that on days of high oxidant exposure, ozone irritated the respiratory systems of cross-country runners sufficiently to restrict inspiratory capacity and to reduce tidal volume. These changes might in turn have prevented the runners from taking up enough oxygen to sustain the work loads they might have sustained on lower-exposure days. (In this case, the work load sustainable by the runners would be inversely proportional to running time.)

Folinsbee's work[15] also suggests that a healthy person exercising submaximally and exposed to ozone levels near the highest ambient concentrations ever achieved in Los Angeles is able to maintain a normal rate of oxygen uptake, even in the face of changes in respiratory pattern and spirometric parameters. Thus, the decrements in running time observed by Wayne in cross-country runners might not have been seen, or might not have been as evident, in shorter-distance runners. Of course, neither the research of Folinsbee nor of Wayne allows conclusions as to the effects of ozone on oxygen uptake in the ill when they are exercising submaximally.

Folinsbee's work has practical significance beyond these interesting physiological considerations. In epidemiologic studies, decrements in health and performance have been quite commonly associated with elevated concentrations of photochemical oxidants as a class. However, the extent to which such decrements might be related specifically to ozone exposure has usually been obscure. Though Folinsbee's studies do not rigorously prove that ozone exposure caused the decreased running times that Wayne observed, they do suggest a plausible mechanism by which ozone alone might have produced the decreased times.

AUTOMOBILE ACCIDENTS

To examine the possibility that oxidant pollution may impair performance, Ury performed a study[61] of the association of automobile accidents with days of elevated hourly oxidant levels. Oxidant levels supplied by the Los Angeles Air Pollution Control District were measured at the District's downtown station. Ury applied two nonparametric analyses, a sign test and a Kendall's tau, to data for each daylight hour of each weekday in the 3-month period from August through October for both 1963 and 1965. Ninety sets of data covering 9 hr daily and 5 days weekly were available for testing. The sign-test results of each set were obtained by taking successive pairs of weeks (first week compared with second; third week compared with fourth, etc.) and scoring a plus (+) if the week with the higher oxidant had more accidents for that set, a minus (-) if it had fewer, and a tie if the accident frequencies or the oxidant levels were equal (Table 10-5).

All accidents involving driving under the influence of alcohol, narcotics, mechanical failures, and spillage on the roadway were excluded from analysis, as were accidents occurring in rain, drizzle, or fog. The author does not state the fraction of total accidents included in these categories, though it may well have been quite large.

In both analyses, photochemical oxidant concentrations correlated positively with the frequency of motor vehicle accidents. The sign test yielded a descriptive two-sided significance level of 4 percent. Kendall's tau yielded a level of 5 percent. In a further analysis, the author compared accident rates on days when hourly oxidant levels exceeded 0.15 ppm to rates on days when levels did not achieve 0.10 ppm. Accident rates were elevated on the higher-pollution days. A Wilcoxon two-sample test yielded a two-sided significance level of 0.07. In this study, oxidant was the only pollutant whose relationship to auto accidents was considered.

TABLE 10-5. SIGN-TEST DATA FOR TESTING THE ASSOCIATION OF OXIDANT LEVELS WITH ACCIDENTS IN LOS ANGELES, AUGUST THROUGH OCTOBER, 1963 and 1965[61]

Weekday	Year	Plus[a]	Minus[a]	Tie[a]
Monday	1963	22	17	6
	1965	18	18	12
Tuesday	1963	26	11	7
	1965	23	19	9
Wednesday	1963	20	15	8
	1965	26	18	10
Thursday	1963	26	12	11
	1965	31	16	5
Friday	1963	27	18	8
	1965	26	19	7
Total	1963	121	73	40[b]
Total	1965	124	90	43[c]
Grand total		245	163	83

[a] Numbers of accidents during pairs of hours at the same time of day on the same day of week, one week apart, were compared. When the number of accidents during the hour with higher oxidant concentration was higher than during the lower-oxidant hour, a +1 was assigned. When the number of accidents in the higher-oxidant hour was lower, a -1 was assigned. Ties occurred when the number of accidents during the 2 hr was identical.

[b] Significantly positive at $\alpha = 0.001$ excluding ties, $\alpha = 0.01$ including ties.

[c] Significantly positive at $\alpha = 0.05$, including or excluding ties.

Ury et al. also reported an extension[63] of the study cited above,[61] in which the effects of both oxidant and carbon monoxide concentrations on motor vehicle accident frequency in Los Angeles were investigated. The investigators assessed the effects of oxidants over the summers of 1963 and 1965 (the same two periods that had been considered in the original study), and they assessed the effects of carbon monoxide over the same two periods and over the winter months of 1964-65 and 1965-66 as well. The same methods of data reduction were used in both studies. In the follow-up study, the sign test was used to correlate the frequency of accidents with mean pollutant concentrations during the hour of the accident and during each of the 3 hr preceding it.

In Ury's second study,[63] none of the 90 individual sets of comparisons specific for day, hour, and year yielded a statistically significant difference between the number of positives and negatives recorded. This is hardly surprising, since no set contained more than six comparisons. However, the degree of significance in the relationship between accident frequency and the oxidant concentration during the hour of the accident was greater than in the original study. This discrepancy between studies arose from a systematic error in data coding, which was discovered between the initial and followup studies. With respect to time of day, the strongest relationships between oxidant concentration and accident frequency were noted at 9 a.m., 10 a.m., and 12 noon. This finding raises the question of whether accident frequency is more strongly associated with an ozone precursor than with ozone itself. With respect to day of week, the strongest relationships were noted on Tuesday and Thursday. No consistent relationship between accident frequency and lagged oxidant concentrations was observed. No relationship between carbon monoxide concentration and accident frequency was observed in any of the periods considered.

Ury et al.[63] present a discussion of variables that might have confounded the observed association between oxidant concentration and accident frequency. It is quite unlikely that inaccuracies in accident reporting or pollutant measurements could have been large enough to influence study results appreciably. Disparities between pollution concentration at accident sites and monitoring sites would have been fairly well absorbed by the statistical methods employed. The study design either negated or minimized effects of hour of day, day of week, and weather. Finally, the lack of association between carbon monoxide concentration and accident frequency argues against an effect of varying traffic density on the results observed. However, the degree to which carbon monoxide measurements actually reflected traffic density must remain open to question, since aerometric information was taken only from a single station.

Possibly other pollutants, particularly nitrogen and sulfur oxides, may have confounded the observed association between oxidant concentration and accident frequency. However, it is unlikely that such other pollutants could have accounted for the bulk of the observed effect, since their concentrations tend to be lower in summer than in winter.

Ury et al. do not speculate as to a mechanism underlying the observed association between oxidant concentration and accident frequency. Conceivably, reduced visual activity, which has been observed by Lagerwerff[26] in humans after a 3-hr inhalation of 392-980 $\mu g/m^3$ (0.2 to 0.5 ppm) ozone, was partly responsible for the observed results. Increased eye irritation[9,46,48,49,54] or reduced visibility during smoggy periods may also have been partly responsible.

VENTILATORY PERFORMANCE IN SOUTHERN CALIFORNIA

The ventilatory performance (measured by the Wright Peak Flow Meter) of two groups of third-grade children living in the Los Angeles Basin was assessed twice monthly for 11 months by McMillan et al.[34] One group of 50 children resided in an area exposed to seasonally high photochemical oxidant concentrations. The other group of 28 children lived in a less polluted area. Aerometric data were obtained from the Los Angeles County Air Pollution Control District. During the 11 months of the study, no correlations between short-term changes in photochemical oxidant pollution and ventilatory performance were observed. Persistently higher ventilatory performance results were obtained from the children residing in the more polluted of the two communities.

The results of this study are difficult to interpret because the two groups of children tested were different in several respects. The children with lower ventilatory performance reported upper respiratory symptoms three times more frequently than the children with higher performance. Also, the majority of children in the less polluted community were from a single ethnic group, whereas children in the other community were ethnically heterogeneous. As closely as we can determine, no statistical adjustment of the data for ethnic differences was made. Furthermore, as noted by the authors, nitrogen dioxide levels were consistently lowest during the winter in the city with elevated oxidant exposures. It is conceivable that the effects of nitrogen dioxide on the study results may have offset those of oxidant. Finally, differences in oxidant exposure in the two towns appear not to have been substantial. During the days of this study, the mean daily oxidant concentration in the low-oxidant city was 0.088 ppm. The corresponding mean in the high-oxidant city was 0.114 ppm.

PULMONARY FUNCTION IN ARIZONA

Lebowitz et al.[27] reported a study of the combined effects of air pollution and weather on the ventilatory function of exercising children, adolescents, and adults in Tucson, Arizona. The study was conducted in the spring and summer of one year.

Ventilatory function tests included the forced vital capacity (FVC) and 1-sec forced expiratory volume (FEV). Tests were administered to four groups of persons living in Tucson. Two of these groups consisted of elementary-school-age children. Since no oxidant measurements during studies of these groups are mentioned in the report, results of these studies will not be presented here.

The third group consisted of 10 white adults of both sexes, aged 22 to 32 years, who walked or ran 5 km (3 miles) along major roads in Tucson during the morning or afternoon of one Saturday in summer. Members of this group appear to have been tested before and after exercising.

The fourth group consisted of nine white adolescents of both sexes, aged 16 to 18 years, who participated in a 32 km (20-mile) walk through major roads in Tucson one Saturday in spring. Members of this group were tested before and after the walk.

Aerometric measurements were obtained from the Pima County Air Pollution Control District and the Arizona State Health Department. Stations used in the studies of adults and adolescents were at least 5 km (3 miles) away from testing sites.

Among adults who performed the 5-km (3-mile) walk or run in the morning, no significant differences between ventilatory function values before and after exercise were observed. During the morning, the average temperature was 28°C (83°F), the relative humidity was 43 percent, and the concentrations of suspended particulate matter and oxidants were, respectively, 104 $\mu g/m^3$ and 0.01 ppm. Among adults who performed the walk or run in the afternoon, post-exercise decreases in ventilatory function of less than 5 percent were observed. These decreases were not statistically significant, and they disappeared within 30 min after exercise. However, they were evidently larger than changes in ventilatory function among those who had exercised in the morning. During the afternoon, the average temperature was 34°C (94°F), the relative humidity was 23 percent, and the concentrations of suspended particulate matter and oxidant were, respectively, 89 $\mu g/m^3$ and 0.03 ppm. On this day, the suspended sulfate level was estimated to have been less than 2.5 $\mu g/m^3$.

Among the nine adolescents who completed the 32-km (20-mile) walk in the spring, consistent and statistically significant post-exercise decreases in ventilatory function were observed. Average values of FVC before and after exercise were, respectively, 3.6 and 3.0 liters. Corresponding average values of FEV_1 were 3.0 and 2.7 liters. On the day of the 32-km (20-mile) walk, the temperature ranged from 15°C to 31°C (59°F to 88°F), the average humidity was 30.5 percent, the average suspended particulate concentration was 133.7 $\mu g/m^3$, the average sulfate concentration was 3.7 $\mu g/m^3$, and hourly peak oxidant concentrations increased with time from 0.01 to 0.12 ppm. The 32-km (20-mile) walk ended about 2 p.m., which is usually near the hour of daily maximal oxidant concentration in Los Angeles. Whether this is also true of Tucson is not stated in the report.

Since oxidant levels appear to have been higher during the 32-km (20-mile) adolescent walk than during the 5-km (3-mile) adult walk or run, and since statistically significant post-exercise decreases in lung function were observed in the adolescents but not in the adults, it is conceivable that hourly maximum oxidant concentrations somewhere between 0.03 and 0.12 ppm may affect the ventilatory function of exercising humans, if exercise is sufficiently strenuous or sustained. However, the Lebowitz study does not provide a rigorous test of this hypothesis, since adolescents and adults underwent different exercise regimens and since the aerometric measurements employed were made at least 5 km (3 miles) from the study site.

Lebowitz et al[27] suggest five hypothetical mechanisms by which pollution might produce decreased ventilatory function: (a) an irritant effect of pollution on the upper airways, (b) bronchial constriction in response to irritation, (c) increase in flow-resistance in the upper airways and possibly in the lower airways, (d) decreases in lung compliance and lung recoil, and (e) transient edema of the alveoli.

Note that two factors other than oxidant exposure may have influenced the outcome of the studies of adolescents and adults. First, fairly substantial levels of suspended particulate matter were present along with oxidants on the days when these studies were conducted. This fact raises the possibility that particulates alone or in combination with oxidants may have affected ventilatory function. Second, the decline in ventilatory function was greater after a 32-km (20-mile) walk than after a 5-km (3-mile) walk or run. The 32-km (20-mile) walk may well have been more demanding for adolescents than the 5-km (3-mile) walk or run for adults, even though the adults were somewhat older. Thus the results might have been observed even in the absence of pollution exposure. These two factors considerably restrict the vigor with which the hypotheses stated in the previous paragraph can be advanced.

EFFECTS OF OXIDANT ON PULMONARY FUNCTION IN JAPAN

Kagawa and Toyama[24] reported a study of the effects of environmental factors on the pulmonary function of 21 children, all aged 11 years, at an elementary school in Toyko, Japan. The study sample appears to have been quite evenly divided by sex. Pulmonary function tests were administered once a week for 29 weeks between June and December 1972. Tests were generally performed between 1 and 3 p.m. Six physiologic variables were measured: total airway resistance (Raw), specific conductance (Gaw/Vtg), maximal expiratory flow at 50 and 25 percent vital capacity left to be expired (respectively, $\dot{V}_{50}$ and $\dot{V}_{25}$), forced vital capacity (FVC), and a pulmonary gas distribution index (GDI) based on the single-breath nitrogen elimination rate following a full inspiration of pure oxygen.

Aerometric measurements were made on top of the children's three-story school. The following measurement methods were used: For oxidant, neutral buffered potassium iodide and coulometric methods; for ozone (O_3), ethylene chemiluminescence; for hydrocarbons (HC), hydrogen flame ionization; for nitric oxide (NO) and nitrogen dioxide (NO_2), Saltzman method; for sulfur dioxide (SO_2), conductimetric method; and for suspended particulate matter (SPM), a light scattering method. Temperature and relative humidity were also measured.

During this study, hourly average concentrations of oxidant ranged from about 0.03 to about 0.17 ppm, and hourly average concentrations of ozone ranged from about 0.01 to about 0.15 ppm. Over this period, the maximum hourly concentrations of NO, NO_2, and SO_2 were about 0.08, 0.23, and 0.05 ppm, respectively. The maximum hourly average concentration of particulate matter was about 350 $\mu g/m^3$.

Simple correlation coefficients were computed between pulmonary function test results over the 29 weeks of study and mean pollutant concentrations (1) between 1 and 3 p.m. on the day of testing, (2) between 12 noon and 1 p.m. on the same day, (3) between 1 p.m. on the day before testing and 1 p.m. on the testing day. Correlations between temperature and humidity (averaged over the same periods) and pulmonary function test results were also computed. A summary of correlation coefficients significant at $\alpha = 0.05$ is presented in Table 10-6. As the table shows, pulmonary function test results were significantly correlated with temperature far more often than with any other environmental factor. Temperature was also correlated positively and significantly with ozone, and negatively and significantly with NO and suspended particulate matter (SPM). Significant correlations between temperature and Raw, $\dot{V}_{50}$, $\dot{V}_{25}$, and GDI were consistently positive. The degree and consistency of correlation of temperature with $\dot{V}_{50}$ and $\dot{V}_{25}$ were particularly striking. Significant correlations of temperature with Gaw/Vtg and with FVC were consistently negative.

The degree of correlation between pollutant concentrations and pulmonary function test results was generally greater with concentrations during testing (1 to 3 p.m.) than during either of the two previous periods. All significant correlations between pollutant concentrations and test results bore the sign that would be expected if pollution

TABLE 10-6. NUMBER OF SUBJECTS FOR WHOM CORRELATION COEFFICIENTS BETWEEN ENVIRONMENTAL AND RESPIRATORY FUNCTION MEASUREMENTS ARE SIGNIFICANT AT $P < 0.05$, JAPAN, 1972[a,24]

Environmental variables		Physiologic variables					
		Raw	Gaw/Vtg	$\dot{V}$ max at 50% FVC	$\dot{V}$ max at 25% FVC	Gas distribution index	FVC
Oxidant	D[b]	1(0.53)[c]	0	0	0	0	1(-0.45)
	1[d]	1(0.49)	1(-0.56)	0	0	0	1(-0.44)
	24[e]	2(0.65~0.63)	0	1(-0.71)	1(-0.60)	0	0
O_3	D	5(0.80~0.45)	5(-0.66~-0.48)	1(-0.62)	1(-0.66)	2(0.56~0.48)	3(-0.69~-0.48)
	1	2(0.66~0.63)	2(-0.68~-0.52)	0	0	0	4(-0.62~0.46)
	24	3(0.62~-0.45)	3(-0.59~-0.45)	0	0	0	2(-0.44)
Hydro-carbon	D	1(0.59)	2(-0.51~-0.44)	1(-0.50)	0	4(0.76~0.56)	4(-0.63~-0.46)
	1	2(0.53~0.48)	1(-0.57)	2(-0.74~-0.56)	1(-0.59)	1(0.55)	5(-0.70~-0.44)
	24	0	0	0	0	0	0
NO	D	0	0	8(-0.56~-0.47)	10(-0.60~-0.47)	0	0
	1	0	0	4(-0.56~-0.49)	7(-0.71~-0.45)	0	0
	24	1(0.69)	1(-0.70)	4(-0.50~-0.44)	7(-0.64~-0.47)	0	0
NO_2	D	0	0	2(-0.51~-0.44)	1(-0.58)	0	0
	1	0	0	3(-0.56~-0.44)	2(-0.68~-0.47)	0	0
	24	0	0	0	0	0	0
SO_2	D	2(0.47)	1(-0.50)	0	1(-0.76)	6(0.55~0.47)	0
	1	1(0.54)	2(-0.53~-0.45)	1(-0.47)	1(-0.75)	1(0.52)	0
	24	0	0	0	0	0	3(-0.59~-0.44)
SPM	D	0	0	1(-0.45)	1(-0.44)	0	0
	1	0	0	0	1(-0.46)	0	0
	24	1(0.58)	0	0	0	0	0
Tempera-ture	D	12(0.72~0.46)	9(-0.76~-0.52)	19(0.82~0.56)	17(0.81~0.46)	3(0.56~0.45)	5(-0.75~-0.47)
	1	13(0.72~0.44)	10(-0.75~-0.44)	19(0.83~0.55)	13(0.81~0.44)	4(0.58~0.44)	6(-0.77~-0.47)
	24	14(0.73~0.48)	9(-0.79~-0.45)	19(0.90~0.58)	19(0.82~0.58)	4(0.61~0.46)	5(-0.75~-0.55)
Relative humidity	D	0	0	0	1(0.50)	0	0
	1	0	0	1(0.46)	1(0.55)	1(0.44)	0
	24	2(0.53~0.45)	3(-0.54~-0.46)	15(0.68~0.44)	12(0.74~0.46)	3(0.55~0.48)	4(-0.74~-0.51)

[a] Total of 21 children.

[b] D, During measurement of respiratory function.

[c] Numbers in parentheses are the range of their correlation coefficients.

[d] One hour before measurement of respiratory function.

[e] 24, Average of hourly values during 24 hr before measurement of respiratory function.

indeed exerted a deleterious effect on pulmonary function. The pollutants most frequently correlated significantly with test results were (in descending order) NO, ozone, HC, and SO_2. A point of interest is that oxidant and NO_2 were only infrequently correlated with test results. In about 25 percent of subjects, the ozone concentration during testing was significantly correlated with Raw, Gaw/Vtg, and FVC. Concentrations of SO_2 were not significantly correlated with pulmonary function parameters as frequently as were concentrations of ozone. However, SO_2 concentrations tended to correlate significantly with the same parameters as ozone. Nitric oxide and NO_2, unlike ozone and SO_2, tended to correlate significantly with $\dot{V}_{50}$ and $\dot{V}_{25}$. On the basis of these findings, the authors suggested that ozone and SO_2 may exert effects primarily in the upper airways, while the nitrogen oxides may exert effects primarily in the lower airways.

Kagawa and Toyama[24] also presented the individual significant correlations between pulmonary and pollution concentrations between 1 and 3 p.m. on the testing day. Such correlations were unusually frequent in three of the 21 students.

In further analysis, the authors statistically corrected for the effects of temperature by computing partial correlations between pollution concentrations (presumably between 1 and 3 p.m.) and pulmonary function test results. This analysis greatly reduced the number of significant correlations between pollution and pulmonary function. However, significant partial correlations of ozone concentration with Raw were observed in three subjects. Significant partial correlations of ozone with Gaw/Vtg and with $\dot{V}_{50}$ or $\dot{V}_{25}$ were observed in one subject each. The pattern of significant partial correlation between SO_2 and pulmonary function remained very similar to that between ozone and pulmonary function. After the partial correlation analyses, only one significant correlation of nitrogen oxides with $\dot{V}_{50}$ or $\dot{V}_{25}$ remained.

Kagawa and his associates continued to collect pulmonary function data from the students described above. Results obtained from November 1972 to October 1973 have been presented by Kagawa et al. in a separate report.[25] In Kagawa's original study, temperature was observed to exert a consistent effect on pulmonary function. To deal with this effect, the investigators divided the study into a low-temperature season lasting from November 1972 to March 1973, and a high-temperature season lasting from April to October 1973. Data were collected on 19 days in the low-temperature season and on 30 days in the high-temperature season. Data from 19 students (10 males and nine females) were analyzed.

As in the first study, simple correlations between levels of environmental factors and pulmonary function test results were computed. The period over which levels of environmental factors were averaged is not clearly stated in the second study. However, we believe that these levels were averaged over the 2 hr between 12 noon and 2 p.m. on the day of pulmonary function testing. In the analysis of the second study, the investigators considered the environmental and pulmonary function variables that had yielded an appreciable number of significant correlations in the first study. The environmental variables considered were temperature, ozone, NO, NO_2, $[NO + NO_2]$, SO_2, and SPM. Oxidant was not considered. The pulmonary function variables considered were Raw, Gaw/Vtg, $\dot{V}_{50}$, and $\dot{V}_{25}$.

During this study, hourly averaged ozone concentrations ranged from about 0.01 to about 0.30 ppm. The maximum hourly average concentrations of NO, NO_2, and SO_2 were about 0.18, 0.30, and 0.16 ppm, respectively. The maximum hourly average concentration of suspended particulate matter was about 450 $\mu g/m^3$.

In the high- and low-temperature season-specific analyses, temperature was consistently positively correlated with Raw, $\dot{V}_{50}$ and $\dot{V}_{25}$, and negatively correlated with Gaw/Vtg. However, when data for one full year were analyzed, temperature was negatively associated with Raw in 17 of 19 subjects. Thus it appeared that the effect of temperature on Raw might be quite heavily dependent of the selection of the study period.

The results of this second study were somewhat similar to those of the first with respect to ozone. Concentrations of ozone were generally positively correlated with Raw and negatively correlated with Gaw/Vtg in both the high- and low-temperature seasons. Interestingly, this was more consistently true during the low-temperature season than during the high-temperature season, when measured ozone concentrations were highest. During the low-temperature season, 8 of the 19 ozone-Raw correlations and 7 of 19 ozone-Gaw/Vtg correlations were significant at $\alpha = 0.05$.

When partial correlation analysis, which statistically removed the effects of temperature, was performed, five ozone-Raw correlation coefficients retained statistical significance. Thus it appeared that the apparent effect of ozone on Raw during the low-temperature season would not have been due to temperature alone. In the high-temperature season, only 1 of 38 correlations of ozone with Raw or Gaw/Vtg was significant. Simple correlations suggested little detrimental effect of ozone on $\dot{V}_{50}$ or $\dot{V}_{25}$ in either season. However, after a partial correlation analysis that incorporated temperature, the number of significant negative correlations between ozone and V_{50} increased from one to five. This result is difficult to square with results of Kagawa's first study,[24] in which correction for temperature considerably reduced the number of significant correlations between pollution and pulmonary function.

As in Kagawa's first study, concentrations of NO were significantly correlated (negatively) with $\dot{V}_{50}$ and $\dot{V}_{25}$ considerably more consistently than with Raw or Gaw/Vtg. However, concentrations of NO_2 were significantly correlated (positively) with Raw and (negatively) with Gaw/Vtg more consistently than with $\dot{V}_{50}$ or $\dot{V}_{25}$. Significant correlations of NO_2 with Raw and Gaw/Vtg occurred only during the high-temperature season. Few significant correlations between SO_2 concentrations and pulmonary function parameters were observed. During the low-temperature season, concentrations of SPM were generally correlated negatively with Raw and positively with Gaw/Vtg. During the high-temperature season, the direction of these correlations was generally reversed. In both seasons, SPM concentrations were generally negatively correlated with both $\dot{V}_{50}$ and $\dot{V}_{25}$. These results suggest quite strongly that the calculated effects of pollution on pulmonary function, as with temperature, may depend quite heavily on the selection of study period.

Among the 19 students in Kagawa's second study, 5 showed significant correlations both between pulmonary function and at least three environmental factors, and between environmental factors and measures of airway resistance (Raw or Gaw/Vtg) as well as maximal flow ($\dot{V}_{50}$ or $\dot{V}_{25}$). Thus the second study, like the first, suggests that some segments of the healthy population may be more susceptible to pollution exposure than others.

Three interesting considerations emerge from the results of Kagawa's studies. First, the results disclose an association between ozone (but not oxidant) and decrements in pulmonary function. Second, even within the healthy population, certain groups may be more sensitive than others to the effects of air pollution exposure. The results of several studies already described in this chapter are consistent with the same hypothesis. One might hypothesize further that such sensitive groups may be at unusually high risk of developing chronic illness after years or decades of elevated pollution exposure. No studies have as yet specifically addressed this hypothesis. Third, statistical techniques currently available do not appear fully equal to the task of separating the health effects of pollutants from temperature or from each other. Available statistical techniques tend to focus the attention on individual environmental factors as separate entities. In reality, however, the effects of an individual pollutant may be dependent both on the presence of other pollutants and combinations of climatic factors. Statistical separation of the effects of individual components of existing environmental conditions may therefore yield a misleading description of the true situation. It is hoped that future advances in statistical methodology will enable investigators to characterize interactions of environmental factors more completely than is possible at present.

DISCUSSION OF THE EFFECTS OF OXIDANTS ON PERFORMANCE

The studies reported by Wayne et al.[65] and by Ury[61] suggest that photochemical oxidant pollution can impair performance of tasks as different as competitive running and automobile operation. As mentioned in the Wayne et al. study, the proportion of runners failing to improve their times increased quite consistently ($r = 0.945$) with increasingly hourly oxidant exposures over a range of 60 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm). However, within individual portions of the oxidant concentration range, unequivocal linear relationship between oxidant exposure and performance could not be discerned.

Findings of Folinsbee's experimental studies of ozone exposures[14,15] suggest that on high-oxidant days, air pollutants may have restricted the mechanical ventilatory function of the runners observed by Wayne et al. to the point where these runners were no longer able to take up enough oxygen to support optimum performance at maximum exertion. Folinsbee's findings further suggest that ozone alone may have accounted for the results observed by Wayne et al. This suggestion remains open to question, since Folinsbee's exposure concentration of ozone, 0.75 ppm, was considerably higher than most oxidant concentrations observed in the Wayne et al. study.

Studies of ambient oxidant exposure and pulmonary function have yielded mixed results. McMillan's study[34] showed no association between either short-term or long-term oxidant exposure and impairment of pulmonary function in children. However, this study is difficult to interpret, since pulmonary function tests were performed at rather wide intervals (twice per month) and since the children in the high- and low-pollution communities were ethnically dissimilar.

The findings of Lebowitz[27] and Kagawa et al.[24,25] in contrast to those of McMillan,[34] suggest that oxidant pollution may contribute to decrements in pulmonary function. The results of Lebowitz, like those of Folinsbee, are consistent with the hypothesis that the degree to which oxidants affect pulmonary function is positively related to the level of exercise undergone by the subjects. Clearly, though, the Lebowitz study must be interpreted cautiously, since its design did not allow for separation of the effects of environmental and meteorologic factors.

The results of Kagawa et al.[24,25] suggest that short-term exposure to oxidant air pollution may affect the large airways more than the small ones. A point of interest is that Kagawa observed stronger correlations of pulmonary function with ozone than with oxidant. Conceivably, the mechanism underlying Kagawa's observations is similar to that underlying Folinsbee's. In any case, the results of Kagawa et al., like those of Wayne et al., enhance confidence that ambient ozone alone exerts effects on humans, whatever the effects of other photochemical substances may be.

Whether transient decrements in pulmonary function constitute a clean-cut hazard to health is not yet known. Recent evidence suggests that childhood infection, often accompanied by impairment of pulmonary function, may predispose subjects to chronic respiratory disease later in life.[28] However, the degree to which pollution-associated impairment promotes such an outcome has not yet been determined.

Eye Irritation In Relation to Variations in Oxidant Levels

PANEL STUDIES

No symptom has been associated with ambient photochemical pollution more frequently or consistently than eye irritation. In an effort to determine the types and concentrations of pollutants responsible for eye irritation, investigators have studied a variety of individuals in the Los Angeles area. Studies conducted in 1954, 1955, and 1956, reported by Renzetti and Gobran[46] of the Air Pollution Foundation, San Marino, California, were among the first studies in this field.

The first part of the Renzetti and Gobran study was conducted from August through November 1954.[46] In this study, several panels of observers were asked to report eye irritation on Tuesdays and Fridays. Later, panelists were asked to report only on those days for which eye-irritating levels of pollution had been predicted. This latter study design may have introduced bias into observed results. In general, the observers were office and factory workers. One of the panels consisted of a group of scientists of the California Institute of Technology. The eye-irritation data were compared with instantaneous values of oxidant concentrations as measured by potassium iodide recorders.

Data from the 1954 study[46] are summarized in Figure 10-7 and in Tables 10-7 and 10-8. These data strongly suggest that the degree of eye irritation increases as the oxidant level increases over a range from nearly zero to about 0.45 ppm. From Figure 10-7, it is impossible to discern a discrete threshold oxidant concentration below which no eye irritation occurs. In Table 10-8, data from a panel of scientists are summarized. The table suggests that oxidant was not the only environmental variable statistically associated with eye irritation in Los Angeles in 1954, although oxidant explained a higher proportion of the variation in eye irritation than any of the other variables measured. Data from the panel of scientists yielded the greatest number of significant correlations between eye irritation and environmental variables other than oxidant.

The second part of the study[46] was conducted from August through November 1955. In Figure 10-8, a regression line relating maximum eye irritation in a panel of scientists to maximum oxidant concentration is presented. The relationship between eye irritation and oxidant concentration is qualitatively similar to that observed in 1954 in that eye irritation increased as the oxidant level increased, and in that a discrete oxidant threshold concentration could not be clearly discerned.

From the data provided by the Air Pollution Foundation studies,[46] linear mathematical relationships between maximum oxidant values and mean maximum eye-irritation values were derived. The data from these studies demonstrated increasing eye irritation with increasing concentrations of oxidant pollution over the range of instantaneous values from 100 to 880 μg/m³ (0.05 to 0.45 ppm), although no clear threshold level for this effect was apparent (Figure 10-7).

Other studies on eye irritation have been performed, including one in which a panel of employees of the Los Angeles Air Pollution Control District was queried during the period 1955-58.[17] A group of environmental sanitation workers in the San Francisco Bay Area was also studied during this same period. These panels demonstrated a tendency to experience increasing occurrence of eye irritation with increasing oxidant levels. As in all such studies, there were some individuals who reported eye irritation even when there was no oxidant present.

TABLE 10-7. CORRELATION OF EYE IRRITATION WITH SIMULTANEOUS OXIDANT CONCENTRATIONS, IN ORDER OF DECREASING EYE IRRITATION SCORE, FOR A NUMBER OF STATIONS IN THE LOS ANGELES AREA, 1954[46]

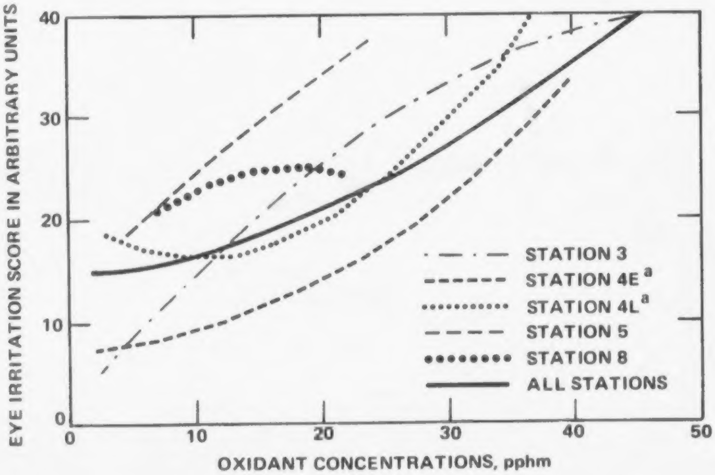
Average eye irritation score	Average oxidant concentration, ppm	Variation in eye irritation score explained by oxidant concentration (r^2)	Station	Number of daily observations
26.2	0.13	0.88	5	25
22.0	0.10	0.68	8	29
21.9	0.21	0.76	4L[a]	24
21.3	0.11	0.06	2	30
18.2	0.15	0.56	3	67
13.0	0.17	0.65	4E[a]	66
18.8	0.14	0.18	11	344

[a] L = panel of laymen in Pasadena, California; E = panel of scientists at the California Institute of Technology in Pasadena.

A large study of student nurses, conducted by Hammer et al.,[18] has been described previously. As mentioned, rates of eye irritation in this study increased with daily maximum hourly oxidant levels in and above the range of 294 to 372 μg/m³ (0.15 to 0.19 ppm).

EVALUATION OF FILTERS FOR REMOVING IRRITANTS FROM POLLUTED AIR

A study was conducted by Richardson and Middleton[48,49] to evaluate the sensory effectiveness of air filter media for removing eye irritants from polluted air. Eye irritation in two



[a] "E"=panel of scientists at the California Institute of Technology. L=panel of laymen in Pasadena, California. For both "E" and "L" panels, aerometric measurements from station 4 were used.

Figure 10-7. Regression curves relating eye irritation scores and simultaneous oxidant concentrations from a number of stations in the Los Angeles area, 1954.[46]

groups of 20 female telephone company employees, similar with respect to age and job characteristics and employed in identical adjacent rooms, was evaluated over 123 workdays from May to November 1956. Active and dummy filter units were switched periodically between the two rooms so that the groups were alternately exposed to test and control conditions. The sensory response of the subjects was measured daily at 11 a.m. by means of a questionnaire; simultaneous measurements of oxidants, particulate matter, and nitrogen dioxide were obtained within each of the two rooms and immediately outside the building.

The differences in eye irritation between the activated-carbon filtered and nonfiltered test situations were in all cases highly significant (Table 10-9). A statistically significant correlation between eye irritation and oxidant concentrations occurred in the nonfiltered room (Table 10-10). The scatter diagram of results (Figure 10-9) suggests that the severity of eye irritation begins to increase above a simultaneous oxidant concentration of about 200 $\mu g/m^3$ (0.10 ppm) as measured by the KI method.

Nitrogen dioxide concentrations were reduced by the activated carbon filters during their early use but, after a period of time, nitrogen dioxide concentrations in the filtered atmosphere increased. No significant correlations between eye irritation and nitrogen dioxide levels were observed, nor were significant correlations found between eye irritation and concentrations of particulate matter.

TABLE 10-8. CORRELATION BETWEEN EYE IRRITATION AND SIMULTANEOUS ENVIRONMENTAL MEASUREMENTS, AS JUDGED BY A PANEL OF SCIENTISTS, 1954[46]

Environmental variables	Variance in eye irritation score explained by environmental variable (r^2)	Average value of variable	Average eye irritation score	Number of observations
Oxidant	0.65	0.17 ppm	1.30	66
NO_x	0.07	0.20 ppm	13.1	51
CO	0.53	0.27 ppm	14.2	47
Hydrocarbons	0.39	0.17 ppm	14.0	53
Visibility	0.17	1.2 miles	13.3	56
Particulates	0.53	21.1 Coh units	13.7	26
Aldehydes	0.48	0.19 ppm	14.0	18

PHOTOCHEMICAL OXIDANT AND EYE IRRITATION IN LOCATIONS OTHER THAN CALIFORNIA

Oxidant measurements at levels possibly associated with eye irritation have been reported from a number of cities other than Los Angeles. Circumstantial evidence of increased eye irritation has been reported in Washington, D.C., Denver, New York City, and St. Louis. An epidemiologic study of eye irritation and other health indices was carried out by Cassell et al.[9] on a population living on the lower East Side in Manhattan. In this study, families reported the presence or absence of symptoms, including eye irritation, each week. In October 1963 (a period of increased pollution) the

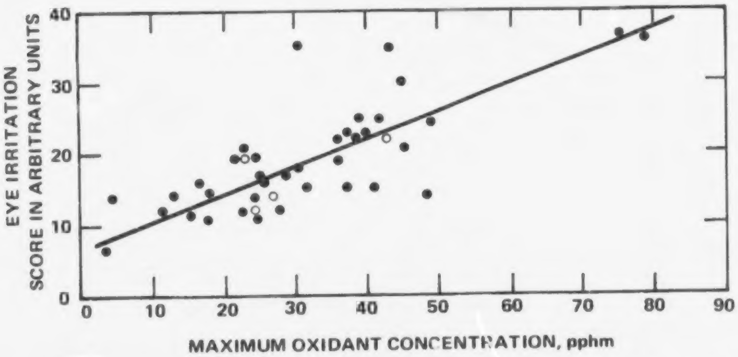


Figure 10-8. Variation of mean maximum eye irritation, as judged by a panel of scientists, with maximum oxidant concentrations, Pasadena, August-November 1955.

frequency of new reports of eye irritation increased from about 2 to nearly 5 percent of the population. However, Cassell's study design did not allow the effects of individual pollutants to be separated from each other.

In the Makino and Mizoguchi study of Japanese students, cited above,[32] the daily frequency of eye irritation regressed on corresponding daily (presumably maximum hourly) oxidant concentration for each of the 12 months of study. Regression lines that yielded significant associations between irritation rate are presented in Figure 10-10. As the figure shows, the rate of eye irritation rose most quickly with increasing oxidant levels during July. The data for that month predicted that 10 percent of students would complain of eye irritation when the oxidant level was about 0.18 ppm, and that 15 percent of students would similarly complain when the level was about 0.26 ppm. On the other hand, data for December predicted that only about 3.5 percent of students would complain of eye irritation when the oxidant level was about 0.26 ppm. Statistically significant associations between oxidant concentration and eye irritation rates occurred more frequently in summer and autumn than in winter and spring.

Shimizu et al.[54] reported a combined epidemiologic, clinical, and toxicologic study of the effects of photochemical smog on the eyes of humans and rabbits. In the epidemiologic portion of the study, all 515 students at a junior high school in Tokyo were requested to complete a symptom questionnaire each day over the 61-day period between May 20 and July 19, 1974. The questionnaire inquired about the presence of 17 symptoms, including eye irritation, eye pain, hyperemia of the eye, lacrimation, blurred vision, and a variety of respiratory and constitutional symptoms.

TABLE 10-9. EFFECT OF FILTER ON SENSORY IRRITATION AND CHEMICAL MEASUREMENTS[49]

Test condition	Eye irritation index[a]	Oxidants, pphm[b]	NO_2, pphm
0.032[c] Activated carbon filter:			
Mean, nonfiltered room	1.99	9.8	1.5
Mean, filtered room	1.01	0.49	0.41
Difference between means	0.98	9.4	1.1
Probability that the difference could have occurred by chance	<0.01	<<0.01	<<0.01
0.016 Activated carbon filter:			
Mean, nonfiltered room	1.95	8.4	3.4
Mean, filtered room	1.41	1.8	1.6
Difference between means	1.54	6.7	1.8
Probability that the difference could have occurred by chance	<0.01	<<0.01	<<0.01
0.075 Activated carbon filter:			
Mean, nonfiltered room	5.45	13.9	2.7
Mean, filtered room	2.35	4.9	5.7
Difference between means	3.10	9.0	3.0
Probability that the difference could have occurred by chance	<0.05	<0.01	[c]
0.0030 Activated carbon filter:			
Mean, nonfiltered room	2.35	7.3	4.7
Mean, filtered room	1.19	3.6	4.9
Difference between means	1.16	3.7	0.2
Probability that the difference could have occurred by chance	<0.01	<0.01	[d]
Particulate filter			
Mean, nonfiltered room	2.13	5.7	6.3
Mean, filtered room	1.91	3.4	5.5
Difference between means	0.22	2.3	0.3
Probability that the difference could have occurred by chance	[d]	<0.01	<0.02

[a] An eye irritation index of 3 corresponds to barely noticeable irritation; an index of 7 corresponds to moderate irritation; an index of 11 corresponds to severe irritation.

[b] Measured by the KI method.

[c] Refers to detention time in seconds.

[d] Difference not significant.

During the 61 days of the epidemiologic study, the weather appears to have been humid and rather cool, with maximum temperatures rarely exceeding 30°C. The oxidant level exceeded 0.15 ppm on only 5 days, and the highest oxidant level recorded during the study was 0.23 ppm. Though averaging times were not specified in the report, these levels appear to have been maximum instantaneous levels. Levels of SO_2 as well as oxidant were presented in the report. The methods used to measure oxidant and SO_2 concentrations were not mentioned.

Symptom rates averaged over the 5 days when oxidant concentrations exceeded 0.15 ppm were compared to corresponding rates averaged over all days of the study. The percentages of students reporting eye symptoms on the high-oxidant days and on all study days, respectively, were as follows: eye irritation, 8.0 and 2.4 percent; eye pain, 3.3 and 1.9 percent; blurred vision, 1.4 and 1.1 percent; lacrimation, 1.4 and 0.5 percent; and hyperemia of the eyes, 0.6 and 0.5 percent. Differences between high-oxidant days and all study days in eye irritation and lacrimation were statistically significant at $\alpha = 0.05$. Incidentally, a significantly higher mean percentage of students also reported sore throat (2.6 versus 1.1 percent) and dyspnea (2.5 versus 1.9 percent) on the high-oxidant days than on all study days. These findings are quite consistent with the findings of Makino and Mizoguchi[32] and Mizoguchi et al.[37]

TABLE 10-10. PEARSON PRODUCT MOMENT CORRELATION OF COEFFICIENTS BETWEEN EYE IRRITATION AND ENVIRONMENTAL FACTORS IN A NONFILTERED ROOM[49]

Item		Factor	Correlation
Irritation	vs.	Oxidants concentration, by phenolphtalein method	0.81
Irritation	vs.	Oxidants concentrations, by KI method	0.31
Irritation	vs.	NO_2 concentrations	0.05
Irritation	vs.	Particulate	0.15
Irritation	vs.	Temperature	0.49
Irritation	vs.	Relative humidity	-0.24
Temperature	vs.	Relative humidity	-0.38
Temperature	vs.	Oxidants concentration, by KI method	0.29
Oxidants concentration, by phenolphtalein method	vs.	Oxidants concentration, by KI method	0.88
NO_2 concentrations	vs.	Oxidants concentration, by KI method	-0.15

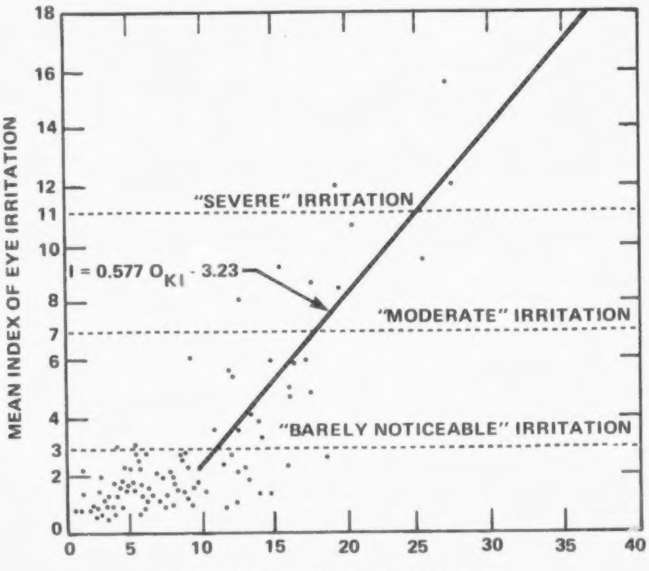


Figure 10-9. Mean index of eye irritation versus oxidant concentration.[49]

Data presented in the Shimizu et al. report[54] suggest that the highest concentrations of both oxidant and SO_2 tended to occur on the same days. Concentrations of SO_2 and oxidant appear to have been of generally similar magnitudes. Thus we do not believe it is justifiable to attribute solely to oxidant the differences between high- and low-oxidant days in symptom rates observed in this study. It is conceivable that the presence of SO_2 or other sulfur oxide compounds may have been a necessary factor in the production of these symptoms.

In the clinical portion of the Shimizu et al. study,[54] the eyes on 41 seventh graders and 41 eighth graders were examined on three sets of 2 consecutive days—one set in May, one in July, and one in October 1974. On the first day of each set, either the seventh or eighth graders were tested. On the next day, the remaining grade was tested. The second day of the 2-day set in July happened to be one of the epidemiologic study days on which the oxidant concentration exceeded 0.15 ppm. On each of the 6 days of the clinical study, subjects were given a physical examination of the eye, the volume and pH of tear fluid were measured, and the amount and activity of tear lysozyme were determined. On the high-oxidant day in July, four (10.0 percent) of 40 students reported symptoms of eye irritation. (This figure is consistent with the 8.0 percent of all students reporting eye irritation on days with oxidant levels exceeding 0.15 ppm.) On the other 5 days of clinical examinations, an average of 1.2 students, or about 3 percent of students examined, reported such symptoms. On the second day of the July clinical examinations, the mean pH of the eighth-graders' tear fluid was significantly lower (about 0.1 pH unit lower) than it had been in the seventh graders tested the day before. On the other two sets of days, when between-days differences in oxidant concentrations were smaller than in July, no significant between-days difference in lacrimal pH was observed. We believe that this finding, though suggestive, must be interpreted cautiously, because different subjects were tested on the 2 days of each set of days. The authors observed no significant pollution-related differences in lacrimal volume or in the volume or activity of tear lysozyme secreted. However, a slight shift in the pattern of lysozyme activity was noted on the high-oxidant day. The significance of this shift is as yet uncertain.

The toxicologic portion of the Shimizu et al. study[54] had two facets. In the first, the eyes of four men and two women aged 20 to 40 years were exposed to 0.5 percent (5000 ppm) acrolein gas. In most subjects, smarting of the eyes developed within 3 min. In others, however, no unusual sensation was noted.

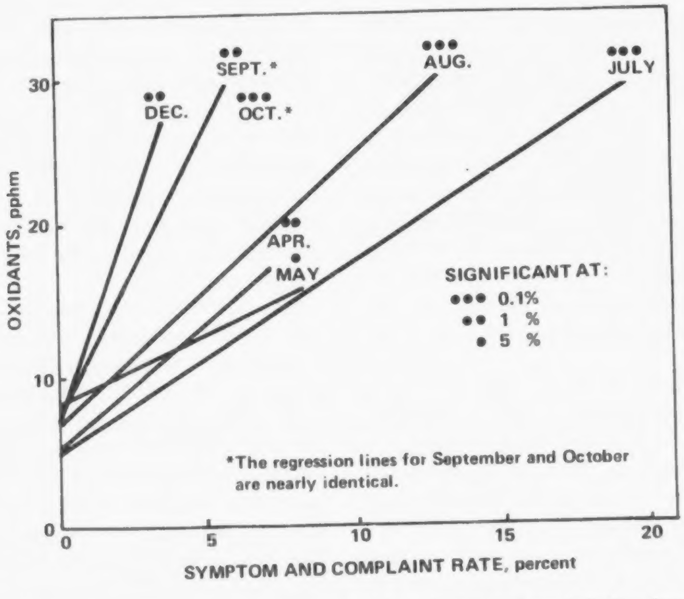


Figure 10-10. Monthly regression lines between oxidant levels and eye irritation (symptom no. 1).[32]

In the second facet of the Shimizu et al. toxicologic study,[54] six rabbits were exposed to 10 ppm acrolein gas for up to 2 hr. One eye of each of two rabbits was kept closed throughout the exposure. After 1 hr of exposure, 3 of 10 eyes had developed spotty, fluorescent stainable, white corneal lesions. After 2 hr of exposure, this type of corneal lesion covered approximately the entire area of the pupil in all the experimental eyes. The authors did not describe the reversibility of these lesions. This type of lesion did not develop in the two closed eyes.

DISCUSSION OF PHOTOCHEMICAL OXIDANTS AND EYE IRRITATION

In the Los Angeles area and in Japan, strong and consistent associations between photochemical oxidant exposures and both the frequency and severity of eye irritation have been observed. In most studies performed to date, the rate of subjects reporting eye irritation has been higher at oxidant concentrations above 0.15 ppm than at lower concentrations. Hammer observed the frequency of eye irritation to increase at maximum hourly oxidant concentrations in the range of 294 to 372 $\mu g/m^3$ (0.15 to 0.19 ppm). Recent Japanese studies, especially that of Makino and Mizoguchi,[32] suggest that eye irritation may begin to occur even at (presumably maximum hourly) oxidant concentrations of 0.10 ppm or below.

As yet, the components of photochemical pollution specifically responsible for eye irritation have not been identified in epidemiologic studies. Such identification must await refinements in available aerometric technology. Experimental studies indicate that ozone, which is the principal contributor to total oxidant concentration, is not itself an eye irritant at ambient levels. However, the possibility remains that ozone in the ambient atmosphere may interact with other substances (which need not necessarily be a part of the photochemical pollutant complex) to produce eye irritation.

Constituents of the photochemical oxidant complex that have been shown to be eye irritants include peroxyacetylnitrate, peroxybenzoylnitrate, acrolein, and formaldehyde. The broad range of actual and potential eye irritants in photochemical smog render it possible, even likely, that different pollutants or blends of pollutants account for eye irritation in different places, or in the same place at different times. This hypothesis can be tested only by further epidemiologic study of pollution-related eye irritation in a variety of locations.

The strong and consistent association observed between short-term photochemical oxidant exposure and eye irritation arouses interest as to what the effects of such long-term exposures on ocular structure and function might be. This concern is heightened by the demonstration by Shimizu et al.[54] of opaque corneal lesions in rabbits resulting from as little as 1 hr of exposure to acrolein. Little epidemiologic effort has as yet been devoted to detecting the ocular effects of chronic ambient oxidant exposures. It is quite conceivable that such effects have not yet become apparent, since no city in the United States has experienced consistently elevated oxidant concentrations for more than 30 years or so. Overall conclusions as to the impact of photochemical oxidant exposures on the human eye must await a thorough, systematic investigation of chronic exposures.

EFFECTS OF CHRONIC PHOTOCHEMICAL OXIDANT EXPOSURES

Introduction

Knowledge of the effects of long-term photochemical oxidant exposure is still distressingly limited. In most studies to date that have assessed such effects, health data have been collected at one point in time in areas whose long-term pollution exposures are expected to have differed. Studies of this design are termed cross-sectional. In carefully planned cross-sectional studies, study populations are selected to be as similar as possible with respect to all characteristics except pollution exposure, lest nonpollution variables interfere with comparisons made among populations. In environmental epidemiology, nonpollution variables of particular importance include cigarette smoking habits, occupational exposure to respiratory irritants, socioeconomic status, and climate.

For two reasons, cross-sectional studies of the effects of long-term pollution exposures must be interpreted cautiously. First, it is virtually never possible to study populations similar in all characteristics except their pollution exposure. Indeed, there is little question that not all the variables complicating comparisons of populations with different pollution exposures have as yet even been identified, let alone controlled in field studies. Second, even when health effects conform to pollution exposure gradients, the investigator, since he has made health measurements at only one point in time, is usually unsure as to the duration or concentration of exposure that may have been necessary to produce the observed effects.

Existing studies must be replicated, or as nearly replicated as possible, before the effects of chronic oxidant exposures can be confidently described. Full characterization of such effects will require carefully designed and patiently conducted prospective studies. As will be seen, these conditions have not yet been fulfilled.

Mortality in Areas of High- and Low-Oxidant Pollution

LUNG CANCER MORTALITY

It is known that active organic carcinogens are found in polluted atmospheres[51] and that ozone, in concentrations above those found in ambient air, has radiomimetic properties.[13] However it is not yet established whether ambient concentrations of organic carcinogens or ozone are sufficient to promote the development of cancer in humans. Buell et al.[5] reported a prospective study of lung cancer among 69,160 members of the California Division of the American Legion. This number represented about 50 percent of all eligible individuals. Since military personnel must meet certain standards of health, the American Legion may under-represent the population with respect to the prevalence of chronic disease. However, it may over-represent the population with respect to cigarette smoking. The cooperating subjects reported by postal questionnaire their residence, occupation, and smoking histories. Identifying data for each individual were maintained on a roster against which the death certificates were checked for the 5-year period 1958-62. It thus became possible to carry out a reasonably economical longitudinal study comparing the major metropolitan areas of California with respect to mortality due to lung cancer and other conditions. A total of 336,571 man-years of observation was included in the report.

As shown in Table 10-11, long-term residents of Los Angeles County had slightly lower age- and smoking-adjusted lung cancer rates than residents of the San Francisco Bay Area counties and San Diego County. These urban groups had higher rates than the population residing in the rest of the state. The relative risk of lung cancer for heavy smokers (more than one pack a day) was greater in Los Angeles County than in other areas of California (Table 10-12). For nonsmokers, the rate in the two metropolitan groups was substantially greater than in the rest of the state. The San Francisco/San Diego rates, however, were higher than those in Los Angeles.

For three reasons, the Buell et al. study must be considered inconclusive. First, the rate of questionnaire return, though impossible to determine precisely, probably did not exceed 60 percent. Thus it is quite conceivable that undetected selective factors may have influenced the published results. Second, if chronic oxidant exposures produce lung cancer, the latent period between the first exposure and tumor development may have exceeded the maximum observation period possible in this study. This maximum period was only about 15 years, between the late 1940's, when Los Angeles first experienced elevated photochemical pollution levels, and 1962. Third, Buell et al. did not relate mortality rates to actual pollution measurements.

CHRONIC RESPIRATORY DISEASE MORTALITY

Mahoney reported a preliminary study[31] in which respiratory disease mortality rates in Los

TABLE 10-11. TOTAL LUNG CANCER MORTALITY IN AN AMERICAN LEGION STUDY POPULATION, CALIFORNIA, 1958-62[5]

	Los Angeles County		San Francisco Bay Area and San Diego Counties		All other Calif. counties	
Item	Mortality rate[a]	Total deaths	Mortality rate[a]	Total deaths	Mortality rate[a]	Total deaths
Age-adjusted[b]	95.9	n/a	104.5	n/a	75.3	n/a
Age- and smoking-adjusted	95.4	n/a	102.0	n/a	75.5	n/a
Residency:[c]						
At least 10 years	96.6	79	106.3	58	19.9	69
Less than 10 years	76.7	27	69.1	13	68.5	30
Unknown	123.4	12	215.3	10	65.2	6

[a] Deaths per 100,000 man-years.

[b] Age-adjusted by the direct method to the total study population.

[c] Age- and smoking-adjusted.

Angeles during 1961 were related to wind-flow patterns in the city. In the Los Angeles Basin, the prevailing wind blows in a southwesterly direction from the Pacific Ocean. Mahoney constructed five concentric zones within the Basin (Zones 1 through 5), each about 10 km (6 miles) wide and each inland and downwind from the last. He then computed respiratory death rates separately for each zone. Death rates were computed by dividing the number of deaths in 1961 by the population in the zone as determined by the 1960 census. Zonal populations ranged from 36,312 in Zone 5, the most downwind and inland zone, to 574,512 in Zone 3. All death rates were adjusted, by the indirect method, for age, sex, and income level. All persons in each zone were assumed to have the median income for that zone. Only whites were considered in the analysis. Variables such as smoking, migration within the city, and variation among zones in population density were not considered.

Adjusted respiratory death rates per 100,000 in Zones 1 through 5, respectively, were as follows: 53, 51, 58, 66, and 111. This finding is consistent with the hypothesis that photochemical air pollution influences mortality rates, since pollution levels are generally higher in leeward areas of Los Angeles than in windward areas. However, as the author mentions, other factors such as temperature and humidity are also intimately associated with wind-flow pattern. The relative contribution to mortality rates of these factors, pollution, and demographic and behavioral variables not considered could not be determined from Mahoney's report. Also, no pollution measurements were presented to document the degree to which pollution levels increased with increasing distance from the ocean. Thus as the author himself observed, this report must be considered inconclusive, suggestive as it is of an effect of pollution on mortality.

Rates of mortality resulting from chronic respiratory diseases other than lung cancer were briefly considered in the Buell et al. study.[5] These rates were somewhat higher in Los Angeles than in San Francisco and San Diego Counties among persons residing for 10 or more years in their respective counties, but the rates were highest in the other less urbanized counties (Table 10-13). Interpretation of these observations is clouded by several factors: (1) The possibility that oxidant pollution may induce chronic respiratory disease only after a long latent period, (2) the lack of actual air pollution measurements, and (3) the lack of control for possible differences in socioeconomic level among the areas compared. Winkelstein et al.[66,67] have observed a strong effect of socioeconomic level on chronic respiratory disease mortality. (In these reports, interarea differences in socioeconomic level may have been minimized by the fact that only American Legion members were studied.)

DISCUSSION OF CHRONIC PHOTOCHEMICAL POLLUTION EXPOSURES AND MORTALITY

Buell et al.[5] observed no consistent association between long-term oxidant exposure and lung cancer mortality in California. Mahoney[31] reported higher total respiratory disease mortality rates in inland, downwind sections of Los Angeles than in coastal, upwind sections. In Los Angeles, oxidant concentrations tend to be higher inland than in coastal areas. Socioeconomic, demographic, and behavioral variables were not fully controlled in either study. Neither were mortality rates related

TABLE 10-12. LUNG CANCER DEATHS AND RELATIVE RISKS PER 100,000 MAN-YEARS IN AN AMERICAN LEGION STUDY POPULATION, BY EXTENT OF CIGARETTE SMOKING AND RESIDENCE, CALIFORNIA, 1958-62[5]

Daily cigarette smoking, lifetime history[a]	Los Angeles County		S. F. Bay Area and San Diego Counties		All other counties[b]	
	Rate	Relative risk	Rate	Relative risk	Rate	Relative risk
None	28.1	2.5	43.9	3.9	11.2	1.0
Less than one pack	63.6	5.7	77.1	6.9	6.10	5.4
About one pack	126.0	11.3	134.5	12.0	124.9	11.2
More than one pack	241.3	21.5	226.0	20.2	137.5	12.3
Ratio:						
$\frac{\text{More than one pack}}{\text{none}}$	3.6		5.1		12.3	

[a] Age-adjusted by the direct method to the total study population

[b] Nonsmokers in all other counties taken as unit risk

to actual pollution measurements. Thus both of them must be considered as inconclusive.

In view of the long latent periods known to be involved in the development of many cancers and chronic diseases, it may be that effects of photochemical oxidant exposures on mortality rates and other health indices have not yet become apparent. Considerable further study, in which the effects of socioeconomic and demographic variables are carefully considered, is required before the true relationship of photochemical pollution to mortality rates can be conclusively defined.

General Morbidity in Areas of High- and Low-Oxidant Pollution

STATE OF CALIFORNIA HEALTH SURVEY

In 1954, weekly surveys of new illness and injury were conducted throughout California by the State Department of Public Health.[6,7,8] Sampling units consisted of homes selected to be representative of all homes in the state. Weekly rates of illness and injury in Los Angeles County were compared to those in the rest of the state during the 17 weeks from August 2 through November 29, 1954. Combined weekly incidence of colds, asthma attacks, hay fever, and other respiratory conditions in persons of all ages are presented in Figure 10-11. As the figure shows, there was little difference between Los Angeles County and the rest of California in average rates of these illnesses over the study period. The high peaks of incidence in October are quite likely reflections of the recent beginning of the academic year, though this is not stated in the report.

In Figure 10-12, weekly incidences of all illness and injury in persons aged 65 and over are presented. As the figure shows, the average weekly incidence of illness and injury was about 10.4 percent in Los Angeles County and about 7.0 percent in the rest of the state. This finding suggests that long-term exposures to photochemical oxidants may promote increased susceptibility to illness in the elderly. However, the finding must be considered inconclusive, since the investigators did not adjust for differences between Los Angeles and the rest of California in such factors as population density, ethnic characteristics, and socioeconomic level.

In Figures 10-11 and 10-12, three high-smog periods in Los Angeles County are noted. Criteria for selection of these periods were not presented in the report. Aerometric data collected during this study in Los Angeles City and Pasadena suggest that during these high-smog periods, maximum concentrations of suspended particulates, airborne lead, and carbon monoxide were generally unusually high. However, maximum concentrations of oxidant and nitrogen dioxide do not appear to have been unusally high during these periods. In any event, weekly incidences of illness in Los Angeles County do not generally appear to have risen appreciably during these periods.

In May and June 1956, another adult health survey was undertaken throughout the state of California.[19,20] One goal of this survey was to determine the prevalence of various conditions in different areas of the state. A probability sample of 3545 households, selected to be representative of all households in the state in the ratio of 1 to 1155, was selected. Populations in service camps and other institutions were not sampled. A questionnaire was administered by personal interview to one adult in each sample household. After weighting for the number of adults in each household, the total study sample consisted of 6939 persons. In this phase of the survey, care was taken not to lead subjects toward attributing their illnesses to air pollution.

Study results were distributed by area of the respondent's residence. In Table 10-14 are presented the proportions of respondents reporting

TABLE 10-13. TOTAL CHRONIC RESPIRATORY DISEASE MORTALITY IN AN AMERICAN LEGION STUDY POPULATION, CALIFORNIA, 1958-62[5]

	Los Angeles County		San Franscisco Bay Area and San Diego Counties		All other counties	
Residency	Mortality rate[b]	Total deaths	Mortality rate[b]	Total deaths	Mortality rate[b]	Total deaths
10 years	33.4	31	28.3	15	45.6	40
Less than 10 years	41.2	14	45.6	8	41.3	17
Unknown	139.1	12	59.8	3	39.7	4
Total	46.7	57	34.0	26	44.4	61

[a] Age- and smoking-adjusted by the direct method to the total study population

[b] Per 100,000 man-years

various respiratory conditions in three counties in the Los Angeles/San Diego area, nine counties in the San Francisco Bay Area, and the remainder of the state. As the table shows, the proportion of respondents reporting cough, nose complaints, and throat complaints was higher in the Los Angeles/San Diego area than elsewhere in the state. The proportion of respondents reporting hay fever was lowest in the Los Angeles/San Diego area. Differences among areas in the proportion of respondents reporting the other conditions listed were slight.

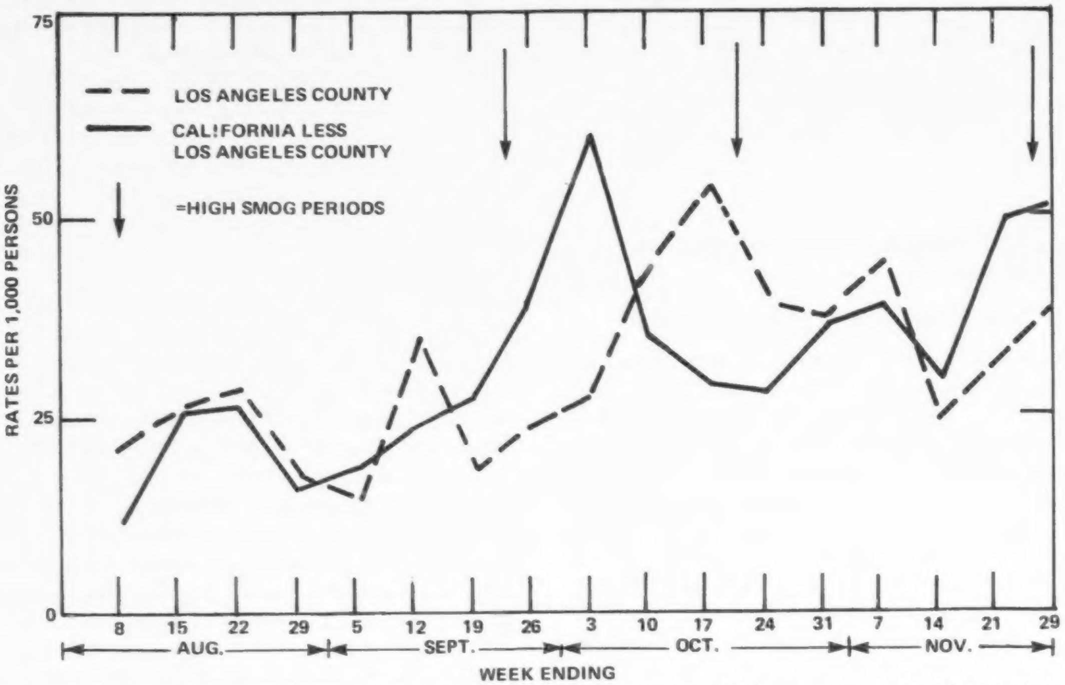


Figure10-11. Combined weekly incidence rates of selected conditions (colds, hay fever, asthma, and other respiratory conditions) in persons of all ages, Los Angeles County and the remainder of California, August 7 to November 28, 1954.[6,7,8]

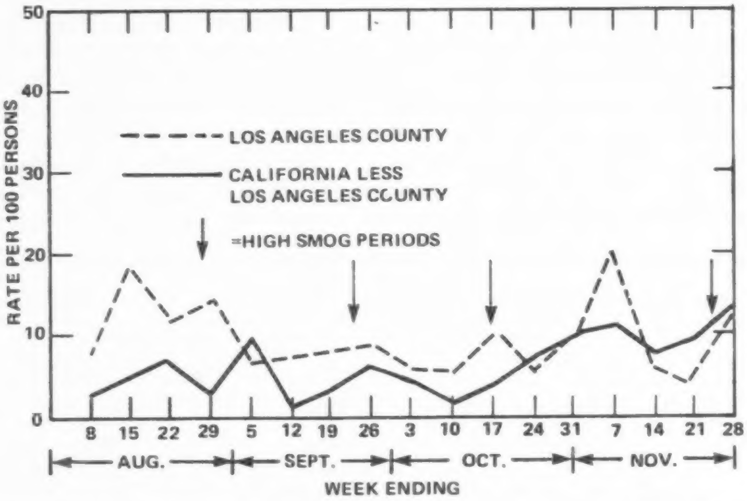


Figure 10-12. Weekly incidence rates of illness and injury for persons aged 65 and over in Los Angeles County and the remainder of California, August 2 to November 28, 1954.[6,7,8]

Another goal of this survey was to determine the degree to which subjects attributed exacerbations of respiratory conditions to air pollution. Figure 10-13 presents the proportions of respondents in Los Angeles County and the San Francisco Bay Area attributing exacerbation of respiratory conditions to air pollution and other factors. As the figure shows, the proportion of respondents attributing exacerbation of each condition to air pollution was higher in Los Angeles than in San Francisco. For all conditions but bronchitis, intercity differences were quite marked. Clearly, it cannot be concluded that air pollution in Los Angeles caused all the exacerbations that respondents attributed to it. However, the findings presented in Figures 10-13 at least indicate that Los Angeles residents considered local air pollution a hazard to health.

A panel consisting of all persons at least 30 years of age who appeared likely to have chronic respiratory disease was selected from the 1956 general population survey described above. Specifically, the panel was selected on the basis of having reported chronic or repeated attacks of bronchitis, asthma, or coughing in 1956. In selecting the panel, the aid of a chronic respiratory disease specialist and of other medical consultants was enlisted. The initial panel consisted of 1070 persons. The panel was interviewed on four occasions--twice in 1957, and once each in 1958 and 1959. Of the 1070 persons in the original panel, 524 were interviewed all four times.

Results of the panel study were reported by Hausknecht.[19] Of the general population sample of 3545 adults interviewed in 1956 (representing a weighted sample of 6939 persons), 41 percent lived in three counties in the Los Angeles/San Diego area, 27 percent lived in nine San Francisco Bay Area counties, and 32 percent lived in the remainder of the state. Of the 524 panelists completing all four interviews in 1957-59, 48 percent lived in the Los Angeles/San Diego area, 25 percent lived in the San Francisco Bay Area, and 27 percent lived in the remainder of the state. Since all persons throughout the state having reported evidence of chronic respiratory disease were selected for the panel, this finding suggests that rates of chronic respiratory disease were disproportionately high in the Los Angeles/San Diego area. Though it is not clearly stated in the report, proportional distributions of bronchitis, asthma, and cough appear to have been very similar in the Los Angeles/San Diego area and in the rest of the state, excluding the Bay Area. In the Bay Area, the proportions of panelists with bronchitis, asthma, and coughing appear to have been, respectively, lower than, similar to, and higher than corresponding proportions in panelists elsewhere in the state. The logical consequence of these finding is that, among the general population, rates of bronchitis and asthma in the Los Angeles/San Diego area appear to have been higher than elsewhere in the state. Rates of cough in the Los Angeles/San Diego area seem to have been higher than in the rest of the state (excluding the Bay Area), and they may or may not have been higher than in the Bay Area.

Three factors substantially cloud the interpretation of Hausknecht's findings. First, many rates necessary to confirm logical inferences are not presented in the report. Second, over half of the original chronic respiratory disease panel did not complete all four interviews. Illness rates among these nonrespondents are not presented. Third, statistical adjustments are not made for

TABLE 10-14. SELECTED RESPIRATORY CONDITIONS REPORTED BY GENERAL POPULATION SAMPLE, CALIFORNIA, MAY 1956[19]

Conditions reported	California		Los Angeles, Orange, and San Diego Counties		San Francisco Bay Area Counties[a]		Rest of state	
	Frequency	Percent[b]	Frequency	Percent	Frequency	Percent	Frequency	Percent
Bronchitis	309	4	156	5	71	4	82	5
Asthma	188	3	104	3	45	2	39	2
Cough	1341	19	746	22	323	17	272	17
Sinus	1202	17	576	17	302	16	324	20
Hayfever	695	10	265	8	221	12	209	13
Nose complaints	751	11	445	13	186	10	120	7
Throat complaints	848	12	505	15	192	10	151	9
Number of persons interviewed	6939[c]	100	3450	100	1846	100	1643	100

[a] San Francisco, Alameda, Contra Costa, San Mateo, Santa Clara, Marin, Napa, Solano, and Sonoma Counties

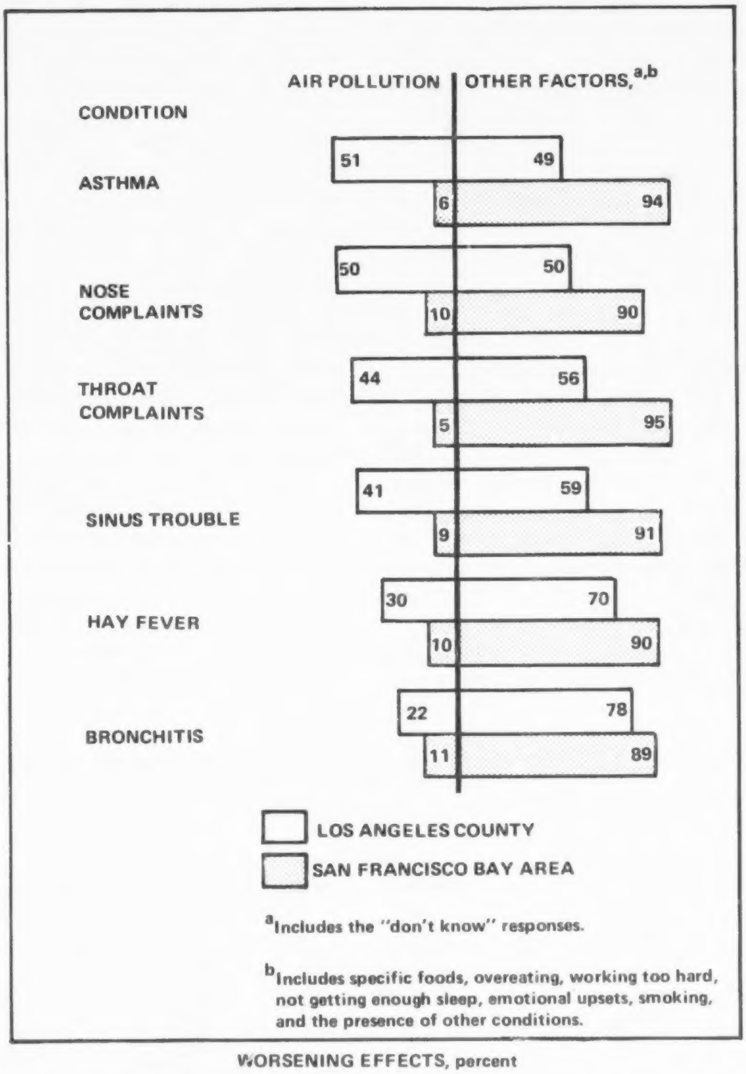
[b] Percentages will not add to 100 because of reports of multiple conditions or of none of the conditions listed

[c] The number of persons interviewed personally was 3545. After weighting for the number of adults in sample households, this figure represented a total sample of 6939

possible interarea differences in the distribution of important covariates (most notably, cigarette smoking habits).

CHRONIC RESPIRATORY DISEASE SURVEY OF TELEPHONE WORKERS

Deane et al.[11] and Goldsmith and Deane[17] used standardized repsiratory illness survey techniques to compare respiratory symptom rates in outdoor telephone company workers in Los Angeles and San Francisco. No aerometric data were presented in these reports. In the older group (aged 50 to 59 years), respiratory symptoms were more frequent in the population of Los Angeles than in that of San Francisco. Persistent cough and phlegm were reported by 31.4 percent of the group aged 50 to 59 years in Los Angeles, compared with 14.3 percent in San Francisco. This difference could not be explained by differences in smoking habits, since in both age groups, proportions of smokers were higher in San Francisco than in Los Angeles. There were no substantial intercity differences in the



[a] Includes the "don't know" responses.

[b] Includes specific foods, overeating, working too hard, not getting enough sleep, emotional upsets, smoking, and the presence of other conditions.

Figure 10-13. Percentage of respondents in Los Angeles County and San Francisco Bay Area attributing exacerbation of respiratory conditions to air pollution and other factors, 1956.[20]

results of pulmonary function tests. In the younger group (aged 40 to 49 years), the frequencies of respiratory symptoms were quite similar in the two cities.

A point of interest is that rates of persistent cough and persistent phlegm in San Francisco were higher in the younger age group than in the older. The proportion of San Francisco smokers was also higher in the younger age group, which may partly explain this finding. However, this is probably not the full explanation, since the younger group also had a higher proportion of smokers in Los Angeles, where symptom rates increased with age. The authors suggested that a lost-to-study factor might have been operating in the older group in San Francisco. Deane and Goldsmith drew no conclusion as to the effect of chronic oxidant exposures on respiratory symptom rates.

Questions regarding eye irritation were also asked by Deane and Goldsmith. The group aged 40 to 49 years in San Francisco reported eye irritation about 10 percent of the time, and the group aged 50 to 59 reported it about 4 percent of the time. The corresponding figures in Los Angeles were 30 and 29 percent, respectively. Perhaps more important is the fact that more than 50 percent of those in both age groups in San Francisco had never experienced eye irritation, whereas the corresponding figure for Los Angeles was less than 10 percent.

OXIDANTS AND EPIDEMIC INFLUENZA

Pearlman et al.[42] studied whether chronic exposure to photochemical oxidants affects susceptibility to influenza infection. These investigators performed a retrospective study during 1968 and 1969 among 3500 elementary school children residing in five southern California communities. Seven to eight hundred second-, fourth-, and sixth-grade students from upper-middle-class socioeconomic areas in each city participated. Selection of these communities was based on historical differences in oxidant exposure, although no difference in exposure was present before or during the epidemic. Pasadena and Riverside were chosen as the high-oxidant cities, Garden Grove as an intermediate city, and San Diego and Santa Barbara as low-oxidant cities. Seasonal peak mean daily maximum hourly oxidant levels recorded in each city from August to October during 1964-67 were, respectively, 451, 368, 235, 157, and 176 $\mu g/m^3$ (0.23, 0.19, 0.12, 0.08, and 0.09 ppm). Concentrations of pollutants other than oxidant in the study cities were not presented in the report.

The incidence and duration of influenza-like illnesses between November 1968 and January 1969 were determined by administration of a questionnaire to the parents of the subjects during March 1969. Parents were asked whether their children had had influenza. If so, the length of illness and presence or absence of fever, coryza, and myalgia were also ascertained. Occurrence of an influenza-like illness was defined as a febrile illness accompanied by coryza and/or myalgia. During April 1969, finger-prick blood specimens were obtained from these children for subsequent titration by hemagglutination inhibition (HI) against A_2/Hong Kong influenza and by complement fixation against type A soluble antigen. Complement fixation established a hemoglobin HI titer of at least 1:32 as indicative of recent infection with A_2/Hong Kong influenza.

In the questionnaire survey, 72.8 percent of the children reported illness, 35.4 percent reported febrile illness, and 21.5 percent reported influenza-like illness. Intercity morbidity did not consistently reflect differences in chronic oxidant exposure. A high- and a low-exposure city (Pasadena and San Diego) reported the greatest degree of febrile illness. Pasadena and Santa Barbara, high- and low-exposure cities, respectively, reported the most influenza-like illness. Adjustment for age and sex differences by probit analysis did not reveal any significant differences in morbidity rates among cities.

Overall, 24.6 percent of children tested were found to have HI titers greater than or equal to 1:32. The percentage of children with significant titers rose from 10.3 percent among those with no illness, to 28.9 percent among those with any illness, to 39.5 percent in those with febrile illness, and to 45.6 percent among those with an influenza-like illness. The frequency of positive titers among febrile and influenza-like illness categories in the two high-exposure cities was lower than that in the two low-exposure cities. This difference was statistically significant. Although the two high-pollution cities studied, Pasadena and Riverside, reported higher subclinical illness rates (elevated titers without illness) than the low pollution cities, the differences were not statistically significant.

Thus Pearlman's study did not demonstrate a positive association between chronic oxidant exposure and influenza-like illness. In animal studies, however, increased morbidity has resulted from acute oxidant exposures. Confirmation of Pearlman's results must await studies in cities exhibiting clear-cut differences in both acute and chronic oxidant concentrations.

Chronic Oxidant Exposure and Pulmonary Function

SURVEY OF INSURANCE COMPANY WORKERS

Linn et al.[29] reported a study in which pulmonary function and respiratory symptom rates in indoor insurance company workers in Los Angeles were compared to those in workers for the same company in San Francisco. Respiratory symptom questionnaires based on the standard questionnaire of the National Heart and Lung Institute were administered to all subjects. Pulmonary function tests included the forced vital capacity (FVC), the 1-sec forced expiratory volume (FEV_1), maximal expiratory flow at 50 percent and 25 percent of vital capacity ($\dot{V}_{50}$ and $\dot{V}_{25}$), closing volume, and slope of the single-breath N_2 alveolar plateau (ΔN_2). Questionnaires were administered in April 1973, and pulmonary function tests were performed in August 1973. In one analysis, sex-specific intercity comparisons of pulmonary function test results were adjusted for age and height. In another analysis, city, smoking, and city-smoking interaction were employed as independent variables. In all, 441 workers in San Francisco and 206 workers in Los Angeles participated in this study.

The proportions of participating males and females were very similar in the two cities, as were the mean heights and weights of men and women. However, the study sample in San Francisco was somewhat older than that in Los Angeles. In San Francisco, 57 percent of the sample was at least 40 years of age; in Los Angeles, the corresponding proportion was 48 percent. Also, higher proportions of both men and women were current smokers in San Francisco (45 percent and 30 percent, respectively) than in Los Angeles (35 percent and 19 percent, respectively). Furthermore, the Los Angeles sample contained a higher proportion of Latin Americans than that in San Francisco, whereas the San Francisco sample contained a higher proportion of Asiatics than that in Los Angeles.

Air pollutant concentrations in the two cities had been measured from 1969 to 1972 at central-city monitoring stations. Over this period, the median oxidant concentration in Los Angeles, expressed according to the post-1974 California standard ultraviolet calibration method, was 0.07 ppm, and the concentration exceeded on 10 percent of days was 0.15 ppm. Corresponding concentrations in San Francisco were 0.02 and 0.03 ppm, respectively. Concentrations of nitrogen dioxide, carbon monoxide, and total suspended particulates were also higher in Los Angeles than in San Francisco. Median concentrations of sulfur dioxide appear to have been very similar in the two cities. Temperature was generally higher and rainfall lower in Los Angeles than in San Francisco, although relative humidity in summer appears to have been slightly higher in Los Angeles.

Sex-specific pulmonary function test results were very similar in the two cities for all tests performed except the ΔN_2 in women, which was higher in San Francisco than in Los Angeles to a degree approaching statistical significance ($p = 0.06$). (Elevations in the ΔN_2, in the absence of other perturbations in pulmonary function, are thought to reflect early obstruction of small airways.) Cigarette smoking was found to have a significant deleterious effect on several indices of pulmonary function in males, and one index (ΔN_2) in females.

Nonpersistent coughing during bad weather at times of day other than the first thing in the morning was reported by 30 percent of women in Los Angeles and by 14 percent of women in San Francisco. Nonpersistent phlegm production under the same circumstances was reported by 23 percent of women in Los Angeles and by 11 percent of women in San Francisco. Respondents had been instructed that high-smog episodes could be considered periods of bad weather. Among women, intercity differences in rates of nonpersistent cough and phlegm were significant at $\alpha = 0.01$.

The results of this study did not reveal a consistent association between chronic photochemical oxidant exposure and impairment of respiratory health, though they suggested an association between oxidant exposure and nonpersistent respiratory symptoms in women. In an interesting discussion, the authors gave several alternative explanations for their findings. First, the effects of acute oxidant exposures may be wholly reversible. Second, oxidant concentrations in Los Angeles may have been offset by other adverse environmental factors such as cold and dampness in San Francisco. Third, oxidant exposures in Los Angeles may actually promote the development of chronic lung disease, but the difference in exposure between Los Angeles and San Francisco may not have been great enough to show such an effect.

STUDY OF SEVENTH DAY ADVENTISTS

Cohen et al.[10] examined the effects of oxidant air pollution on pulmonary function in nonsmoking adults. Seventh Day Adventists were chosen for study, since few of them smoke, and they constituted a readily accessible population that was interested in health. The San Gabriel Valley in California was selected as the high-pollution area, and the San Diego area was chosen as a low-pollution area.

The study population in each area consisted of white English-speaking adults aged 45 to 64 who had not smoked in the last 20 years and who had a lifetime history of smoking less than one pack per year. Data were collected in January 1970. Each participant was interviewed by a physician and underwent tests of ventilatory function, including the 1-sec expiratory volume (FEV_1) and the forced vital capacity (FVC). Study participants were also requested to complete a respiratory questionnaire that was a modified combination of the British Medical Research Council respiratory questionnaire and the London School of Hygiene cardiovascular questionnaire. Aerometric data were obtained from the National Air Pollution Control Administration, which made measurements during September 1969 and January 1970. Daily oxidant measurements for January 1970 were also obtained from the Los Angeles County and San Diego County Air Pollution Control Districts. In addition, historical records from the Air Pollution Control Districts of these two counties were reviewed to obtain an estimate of chronic exposures.

Average daily maximum hourly oxidant levels in the San Gabriel Valley and in San Diego during the month of study were 235 and 137 $\mu g/m^3$ (0.12 ppm and 0.07 ppm), respectively. Measurements of total suspended particulates, respirable particulates, and sulfur dioxide were essentially equivalent in both areas during the time of study. From 1963 through 1967, both areas experienced similar arithmetic mean oxidant concentrations (0.047 ppm in the San Gabriel Valley and 0.038 ppm in San Diego). However, over the same period, the mean of daily maximum hourly oxidant concentrations in the San Gabriel Valley (0.144 ppm) was nearly twice as high as the corresponding mean of 0.074 ppm in San Diego. Also, the proportion of days in this period on which daily maximum hourly oxidant concentrations in the San Gabriel Valley exceeded 0.15 ppm (44.8 percent of days) was about seven times higher than the corresponding proportion of days (6.1 percent) in San Diego. Average concentrations of other pollutants (carbon monoxide, nitric oxide, nitrogen dioxide, hydrocarbons, sulfur dioxide, and particulates) during 1963-67 were generally higher in the San Gabriel Valley than in San Diego.

The prevalence of respiratory symptoms did not differ significantly between the two areas. Prevalence rates generally were considerably lower than those observed in most other studies. Similarly, no significant differences were found in measurements of pulmonary function between areas, and all measurements were within expected normal values.

Thus Cohen et al.[10] demonstrated no significant difference in symptom rates or pulmonary function between the San Gabriel Valley and San Diego. Interpretation of this study is clouded by the similarity of annual average exposure levels between the two areas. Clarification of the question of whether ambient oxidant exposure affects pulmonary function and respiratory symptom rates must await studies in areas having unequivocally different acute and chronic exposure levels.

DISCUSSION OF CHRONIC OXIDANT EXPOSURES AND PULMONARY FUNCTION

As yet, no association between chronic ambient oxidant exposure and pulmonary function has been observed. However, as with other indices of chronic exposure, very few studies investigating such a relationship have yet been performed. Furthermore, those that have been performed are limited in their import, because oxidant exposures for the test population cannot be known with certainty and because complicating effects of other personal and community environmental factors cannot be reliably evaluated.

We have been unable to find any studies of long-term exposure effects on the pulmonary function of those whose health is impaired. Also, no prospective studies have yet been undertaken to determine whether chronic exposures promote declines in the pulmonary function of once-healthy individuals. Finally, effects of long-term oxidant exposure on pulmonary function, as on other indices of chronic exposure, may only now be starting to become evident.

ATTITUDES OF LAYMEN AND PHYSICIANS TOWARD OXIDANT AIR POLLUTION

State of California General Health Survey

At the end of each interview in the general health survey undertaken in California in 1956 and previously described,[20] direct questions concerning the effects of air pollution were asked. Seventy-five percent of the whole surveyed population in Los Angeles County was bothered by air pollution, in contrast to 24 percent in the San Francisco Bay Area and 22 percent in the rest of the state. Corresponding proportions among the working population were, respectively, 80, 29, and 27 percent (Table 10-15). Thirty-two percent of native-born Californians were bothered by air pollution, and 39 percent of those moving to California were so bothered. Of those who were bothered by air pollution, 17 percent in Los Angeles considered moving because of it, in contrast to 4 percent in San Francisco and 12 percent in the rest of the state. Of the same total number, 9 percent in Los Angeles considered changing jobs because of air pollution, in contrast to 3 percent in both San Francisco and the rest of the state. About 20 percent of the state residents who had moved out of a polluted area reported that pollution had some influence on their decision to move; 4 percent gave air pollution as their sole reason for moving. Among those who moved from California communities because of air pollution, 75 percent had moved out of Los Angeles County, 8 percent had moved out of the San Francisco Bay Area, and 17 percent had moved out of other areas of the state. Air pollution was given as the reason for 13 percent of the moves from Los Angeles County since 1947. In other areas of the state, the proportion of moves attributed to air pollution was 1 percent or less.

Eye irritation was the most frequently reported effect of air pollution. In some instances, this symptom was accompanied by nasal irritation (Table 10-16). In metropolitan areas, 80 percent of respondents bothered by air pollution complained of eye irritation. In nonmetropolitan areas, the corresponding proportion was 29 percent.

Before air pollution was specifically mentioned by interviewers, respondents were asked several questions about satisfaction with the communities in which they lived. About 21 percent of all respondents in Los Angeles County expressed dissatisfaction with the communities in which they lived, as compared to 18 percent in both the Bay area and the rest of the state. A far greater proportion of dissatisfied Los Angeles residents (32 percent) voluntarily attributed their dissatisfaction to air pollution than did residents of the San Francisco Bay Area (1 percent) or those in the rest of the state (6 percent).

TABLE 10-15. PERCENT OF SURVEY RESPONSE OF GENERAL AND WORKING POPULATION BOTHERED BY AIR POLLUTION, BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956[20]

Responses	California	Los Angeles County	San Francisco Bay Area	Rest of state
General population sample	6393	2892	1846	2210
% Not bothered by air pollution[a]	55	24	76	78
% Bothered by air pollution:				
Either at home or work	45	75	24	22
Both at home and at work	14	27	4	4
At home only	24	39	14	13
At work only	7	8	6	5
Total % at home	38	66	18	17
Total % at work	21	35	10	9
Working population sample	3732	1577	1028	1127
% Not bothered by air pollution	51	20	71	73
% Bothered by air pollution:				
Either at home or work	49	80	29	27
Both at home and at work	25	49	7	9
At home only	12	16	11	8
At work only	12	15	11	10
Total % at home	37	66	18	17
Total % at work	38	65	18	18

[a]Percents are rounded independently.

Survey of Los Angeles Physicians

A survey of Los Angeles physicians was conducted jointly by the Los Angeles County Medical Association and Tuberculosis and Health Association in December 1960.[30] A sample representing about one in 16 of those physicians registered to practice in the county during 1958 was drawn, resulting in a sample of 526 from a total of 9228 physicians. A pretested questionnaire was mailed with a letter signed by the chairman of the air pollution subcommittee. A follow-up was also mailed, and telephone calls were made to the offices of those physicians who had not responded.

Of the questionnaires mailed, 350 were returned. Of those, 307 (58 percent of the original sample of 526) were completed and tabulated. The words "air pollution" did not appear in the questionnaire, although bias could have been introduced by the fact that the chairman of the air pollution subcommittee attached a letter to the questionnaire. Seventy-seven percent of the physicians who returned completed questionnaires believed that air pollution adversely affected the health of their patients. Two-thirds of the responding physicians felt that air pollution was a factor adversely affecting chronic respiratory disease. One-third of the physicians had advised one or more of their patients to leave the Los Angeles area for health reasons; air pollution was a factor mentioned in two-thirds of these instances. By extrapolation from the sample, assuming it to be representative, it was estimated that physicians had advised more than 10,000 patients to move. It was reported that approximately 25 percent of the patients had done so. Nearly one-third of the physicians had themselves considered moving from the Los Angeles area because of air pollution. Among other environmental factors mentioned as deleterious to health were overcrowding and traffic congestion, but these factors were not mentioned nearly as often as air pollution.

Discussion of Attitudes of Respondents and Physicians Toward Oxidant Air Pollution

The studies summarized in this section (10.4) were subjective in nature and did not yield scientifically confirmed information about rates of illness or functional impairment associated with oxidant pollution. However, the results presented here are interesting in that they demonstrate considerable concern, on the part of laymen and physicians alike, about the effects of such pollution.

SUMMARY OF EPIDEMIOLOGIC APPRAISAL OF PHOTOCHEMICAL OXIDANTS

In this summary, a brief discussion of findings in the preceding three sections of this chapter will be presented (Effects of Short-Term Photochemical Oxidant Exposures, Effects of Chronic Photochemical Oxidant Exposures, and Attitudes of Respondents and Physicians Toward Oxidant Air Pollution). The discussion of each section will begin with the health indices that can be most confidently associated with ambient oxidant exposures and will work toward those indices for which little association with such exposures has been shown. Directions for future research will also be identified.

TABLE 10-16. AIR POLLUTION EFFECTS REPORTED IN GENERAL POPULATION SURVEY, BY TYPE OF COMMUNITY AND BY MAJOR GEOGRAPHIC AREAS IN CALIFORNIA, MAY 1956[20]

Item	Total California		Los Angeles County		San Francisco Bay Area		Rest of state	
	At home	At work	At home	At work	At home	At work	At home	At work
General population sample	6939	3732	2392	1577	1846	1028	2201	1127
Respondents bothered by air pollution	2616	1410	1904	1012	326	190	386	208
% Bothered by air pollution	38	37	66	64	18	18	17	19
	% Persons bothered							
Air pollution effects cited:								
Eyes, effects	75	76	89	88	38	39	41	51
Eye irritation	44	46	54	53	17	18	22	30
Eye and nasal irritation	23	24	26	27	15	14	14	19
Eye irritation and annoying	5	3	6	4	4	3	3	2
Eye, nasal irritation and annoying	3	3	3	4	2	4	1	-
Nasal irritation, eye not mentioned	10	9	5	4	22	23	22	21
Nasal irritation	8	8	4	3	19	19	18	21
Nasal irritation and annoying	2	1	1	1	3	4	4	-
Annoying only	5	7	2	3	17	26	10	8
Other effects only	5	2	2	1	5	5	17	4
No effects reported	5	6	2	4	18	7	10	16
Total %	100	100	100	100	100	100	100	100

Effects of Short-Term Photochemical Oxidant Exposures

The health index most frequently and consistently associated with short-term exposures to ambient photochemical oxidants is eye irritation. All available evidence suggests that ozone alone is not an eye irritant at ambient concentrations. However, the possibility that ozone may interact with other substances to produce eye irritants remains open. In American studies, daily maximum hourly oxidant concentrations above which the rates and severity of eye irritation have been observed to increase have ranged from about 200 $\mu g/m^3$ (0.1 ppm) to about 294 $\mu g/m^3$ (0.15 ppm). Recent Japanese studies raise the possibility that even lower oxidant concentrations may promote eye irritation under certain conditions. The consistency of the association between short-term exposures and eye irritation arouses interest as to what the ocular effects of long-term exposures might be. Until careful studies of long-term exposure effects on the eye have been performed, the question of whether transient instances of eye irritation constitute bona fide impairments of health must remain open.

Short-term photochemical oxidant exposures have been quite consistently associated with decrements in human performance. Wayne et al.[65] have observed the proportion of high school cross-country runners failing to improve their running times to increase as hourly oxidant concentrations increased from about 59 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm). However, in the range of 0.03 to 0.10 ppm oxidant, no consistent linear relationship between oxidant concentration and performance could be detected. Folinsbee's experimental studies suggest that on high-oxidant days, the irritant effect of pollutants may have restricted the runners' mechanical lung function sufficiently to prevent them from taking up enough oxygen to support the performance level of which they were potentially capable. The studies of Folinsbee et al.[14,15] suggest further that ozone alone may have been responsible for this effect.

Ury et al.[63] have reported a statistically significant positive association between hourly oxidant levels and automobile accident frequency in Los Angeles. The study design employed by Ury did not allow dose-response relationships to be developed. Reduced visual acuity, reduced visibility, and eye irritation may all be contributing factors to Ury's findings.

The observations of Lebowitz et al.[27] suggest that the degree to which photochemical oxidants affect pulmonary function may depend quite heavily on the subject's level of exercise. This observation is quite consistent with the experimental findings of Bates[1] and Folinsbee et al.[14,15] It must be noted that too few pollution measurements were reported by Lebowitz to support a dose-response relationship. Also, the Lebowitz et al. study design did not permit oxidant effects to be separated from the effects of other pollutants and of meteorologic factors.

Kagawa and Toyama[24,25] observed that impairment of pulmonary function in Japanese schoolchildren was more strongly associated with exposure to ozone than to total oxidants. This observation enhances confidence that ozone alone may be responsible for decrements in pulmonary function, no matter what the effects of other pollutants may be. Kagawa also observed larger correlations of ozone concentration with indices thought to reflect large airway function (airway resistance and specific conductance) than with indices thought to reflect small airway function (instantaneous flow at 25 and 50 percent of vital capacity). The Kagawa and Toyama study design did not allow dose-response relationships to be inferred.

Several investigators have noted associations between short-term oxidant concentrations and the frequency of respiratory and other symptoms in healthy people. In their large study of student nurses, Hammer et al.[18] observed simple frequencies of cough and chest discomfort and the adjusted frequency of headache to increase with daily maximum hourly oxidant concentrations in and above the range of 588 to 764 $\mu g/m^3$ (0.30 to 0.39 ppm). Japanese investigators have observed the frequencies of several symptoms in schoolchildren, including sore throat, headache, cough, and dyspnea, to be higher on days when maximum hourly oxidant concentration equalled or exceeded 0.15 ppm than on days when corresponding concentrations were below 0.10 ppm. In Japanese studies, symptom frequencies have generally been more strongly associated with total oxidant concentrations than with ozone concentrations. Results of Japanese studies also suggest that people with allergic tendencies and orthostatic dysregulation are more susceptible to short-term photochemical oxidant exposure than are other segments of the population.

Japanese studies have generally shown oxidant- or ozone-associated effects at lower measured oxidant or ozone concentrations than American studies. The consistent difference between Japanese and American findings raises several questions, including whether the components of oxidant pollution in Japan are different from those in Los Angeles, or, indeed, from those in any U.S. location. It is possible, for instance, that oxidants in Japan are accompanied by higher sulfur oxide levels than in the United States. In any case, the Japanese results cited in this chapter underscore the point that epidemiologic results gathered in one area can be generalized to other areas only with the greatest caution. The Japanese results also demonstrate the need to gather comprehensive data in U.S. locations other than Los Angeles.

As yet, it is not possible to judge confidently whether transient symptoms, irritation, or decrements in pulmonary function constitute bona fide impairments of health. As with eye irritation, confident judgement must await studies of the cumulative effect of many of these minor insults occurring over long periods of time.

Short-term photochemical oxidant exposures have also been associated with aggravation of existing disease, though not as reliably as with the production of symptoms and minor illness in the healthy. Schoettlin and Landau[53] observed a significantly higher rate of asthma attacks on days when the (presumably maximum hourly) oxidant concentration exceeded 0.25 ppm than on days when it did not. The results of Schoettlin and Landau also suggested that a portion of the asthmatic population might be particularly susceptible to oxidants.

Motley et al.[38], Remmers and Balchum,[45] and Ury and Hexter[62] have all observed a beneficial effect of air filtration on the lung function of patients with chronic respiratory disease. In studies reported by these investigators, changes in pulmonary function appear to have been more strongly correlated with changes in oxidant concentrations than with changes in nitric oxide or nitrogen oxide concentrations. However, these studies do not support confident estimates of dose-response relationships. Also, these studies are open to fairly serious methodologic questions.

Studies of the relationship between short-term oxidant exposures and hospital admissions have yielded mixed results. Although Brant and Hill[2,3] and Sterling et al.[59,60] have observed positive associations between exposures and admissions, their observations must as yet be considered inconclusive for at least three reasons. First, these investigators have not ruled out an association between nonoxidant pollutants and admissions that might be just as strong as that between oxidant and admissions. Second, the Brant and Hill observation of high positive correlations between oxidant concentrations and admissions 4 weeks later is difficult to rationalize pathophysiologically, particularly in view of the negative correlations occurring in the intervening time. Third, coefficients of correlation between oxidant concentration and same-day admissions have not exceeded the relatively small value of 0.27 (r^2 = 0.073).

As yet, no convincing association has been shown between short-term oxidant exposures and rates of mortality resulting from any cause. The positive association observed by Mills[36] may yet prove to be valid. However, the vigor with which his conclusions can be advanced is considerably limited by the reanalysis by Breslow and Goldsmith[4] of a considerable portion of the data used by Mills, in which no association between oxidants and mortality was observed.

Interpretation of mortality studies to date has been hampered by limitations in statistical methodology. It has not yet proven possible to separate fully the effects on mortality of oxidants, other pollutants, and meteorologic factors, most notably temperature. Future improvements in statistical methodology may allow these factors to be separated more clearly than they can be at present. Future biomedical research should also enhance knowledge of the combined effects of pollution and meteorologic factors. Only with such advances can the true relationships of oxidants to mortality be determined.

Effects of Chronic Photochemical Oxidant Exposures

The effects on human health of long-term photochemical oxidant exposures have not been characterized nearly as completely as those of short-term exposures. When impairments in health have been associated with long-term exposures, it has not yet proven possible to determine the level or duration of exposure necessary to promote the impairments, since most studies of long-term exposure effects have been cross-sectional in design, not longitudinal. Also, studies of long-term exposure effects, whether or not they have revealed associations between exposure and health impairment, have often been rather limited with respect to methods of data collection or analysis. Furthermore, since no American city has experienced elevated oxidant levels over more than about 30 years, consistent effects of long-term exposures conceivably have gone undetected before the present time, no matter how carefully studies were designed and conducted.

Several studies suggest a tenuous association between long-term oxidant exposures and chronic respiratory morbidity. Deane and Goldsmith[11] have observed higher rates of persistent cough and phlegm in Los Angeles telephone workers aged 50 through 59 years than in comparable workers in San Francisco. Hausknecht has reported that a disproportionate number of chronic respiratory disease patients, randomly selected from the whole state of California, lived in the Los Angeles area. Linn has observed higher rates of non-persistent cough and phlegm in women living in Los Angeles than in San Francisco, a finding that would reflect an effect of oxidant on chronic or acute respiratory morbidity. However, the findings of these studies are not clear with respect to the existence of an association between oxidant exposure and chronic respiratory disease. Nor do the findings allow confident inference as to the level or duration of oxidant exposure necessary to promote increased rates of chronic respiratory illness.

Buell et al.[5] observed no consistent association between long-term oxidant exposure and lung cancer mortality in California. Mahoney[31] has reported higher total respiratory disease mortality rates in inland, downwind sections of Los Angeles than in coastal, upwind sections. However, limitations in statistical control for important covariates and a lack of actual pollution measurements render the results of both studies inconclusive.

In view of the long post-exposure latent periods known to be involved in the development of cancer and other chronic diseases, it is quite conceivable that an influence of chronic oxidant exposure on mortality could go undetected until now or sometime in the future. Studies conducted carefully over the next decade or so may well provide the most useful information concerning the relationship of chronic oxidant exposure to mortality.

As yet, no association between chronic oxidant exposure and acute illness has been shown. As far as we are aware, the Pearlman et al. study[42] is the only one available in this field.

Linn et al.[29] and Cohen et al.[10] have reported epidemiologic studies of chronic oxidant exposures and pulmonary function. In neither study has an association between such exposure and pulmonary function been observed. However, interpretation of these results is limited by imperfect matching of high- and low-exposure populations, incomplete knowledge of actual exposure levels, and the relative absence of individuals likely to be at high risk in the study populations (e.g., those with clear-cut chronic respiratory disease).

Attitudes of Laymen and Physicians Toward Oxidant Air Pollution

Studies of community attitudes toward oxidant pollution do not yield scientifically confirmed information regarding the effect of oxidant pollution on illness rates and physiologic function. However, these results are interesting in that they demonstrate considerable community concern about the effect that oxidant pollution may be exerting on the public health.

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11. EFFECTS OF PHOTOCHEMICAL OXIDANTS ON VEGETATION AND CERTAIN MICROORGANISMS

INTRODUCTION

Injury to terrestrial vegetation was one of the earliest manifestations of photochemical oxidant air pollution.[2,8,9,21,22,24,100,144,160,199,200,201,203,212,216,217,219,220,238,263,266,273,274,300,314] Investigations by Middleton et al.[203] in 1944 described smog-induced injury to leafy vegetables, ornamentals, and field crops in a small area of Los Angeles County. By 1950, such injury was observed over a large portion of southern California and the San Francisco Bay Area.[199] Plant injury caused by photochemical oxidants now occurs commonly in most, if not all, of the metropolitan areas of the United States. With increasing frequency, photochemical oxidants are associated with injury to vegetation in rural areas far removed from urban complexes.

Analysis of the photochemical oxidant air pollution complex has resulted in the isolation of three specific phytotoxic components: ozone, nitrogen dioxide, and peroxyacylnitrates. The peroxyacylnitrates are the most phytotoxic of the known photochemical oxidants. A homologous series of compounds, they include peroxyacetylnitrate (PAN), peroxypropionylnitrate (PPN), peroxybutyrylnitrate (PBN), peroxyisobutyrylnitrate ($P_{iso}BN$), and peroxybenzoylnitrate (PBzN). The degree of phytotoxicity of these compounds increases with the increase in their molecular weight: $PAN < PPN < P_{iso}BN < PBN$ (PBzN has not been tested).[212,217,300] Because PPN and PBN are not usually detected in the atmosphere, PAN is the only member of the series that has received attention. Discussion of the peroxyacetylnitrates will therefore be restricted to PAN. The phytotoxic characteristics of nitrogen dioxide will be discussed in a separate document. Consideration of the specific effects of ozone and PAN will be limited to laboratory and controlled field exposures, since quantitative effects of these compounds cannot be easily differentiated in the ambient environment. The term "oxidant" will therefore be used exclusively when discussing ambient air photochemical oxidant exposures that contain variable combinations of PAN, O_3, NO_2, and, in most cases, additional phytotoxicants.[100,116,133,217] Ozone, the most prevalent photochemical oxidant, has received the greatest amount of study, and its effects on vegetation are best understood. The first studies calling attention to ozone as a pollutant affecting crops were the studies of Richards et al.[250] using grapes, and Heggestad and Middleton[125] on tobacco.

Interactions of the oxidant mixtures and other atmospheric contaminants may produce synergistic effects in many plant species[10,68,191,216,217,238,245,269,288] and antagonistic effects on others.[146,292,293] Increasing attention is now being focused on these effects. Sensitive vascular plants are useful biological indicators of photochemical air pollution.[21,22,44,116,120,149,197-199,201,202,204,212,216,217,219,220,221,238,255,266,300,301]

The limited knowledge relating to the direct effects of photochemical oxidants on nonvascular plants and microorganisms is also discussed in this chapter. Although some mosses and lichens may be useful as biological indicators of sulfur compounds, there is no evidence indicating that these nonvascular plants are highly sensitive to photochemical oxidants.

VASCULAR PLANT RESPONSE TO PHOTOCHEMICAL OXIDANTS

The effects of photochemical oxidants on vascular plants can be envisioned as occurring at several response levels, from the molecular to the organismal (Figure 11-1), depending on the concentration of pollutant, length of exposures, and elapsed time between the exposure and the observation of the effects. The earliest effects include an increase in cell membrane permeability, a decrease in carbon dioxide fixation, and a stimulation of stress-induced ethylene production.[113,283,294] These subtle cellular changes are followed by both inactivation and/or activation of specific enzymes, alteration in metabolite pools, and modified metabolite translocation.[283,286,295] Biochemical changes in individual plants are ultimately expressed in visible foliar injury, premature senescence and increased leaf abscission, reduced plant vigor and growth, and death. In the final analysis, biochemical modifications on an individual level are manifested by changes in plant communities and, ultimately, in whole ecosystems.[217,283] These changes occurring in stressed systems can ultimately be measured in socioeconomic impacts. The sequence of topics in this chapter describing photochemical oxidant effects on vascular plants is based on the logical hierarchical ordering of plant response depicted in Figure 11-1. The complexities of the entire subject are apparent in the sections on dose response and factors affecting plant response.

Physiological Processes

In vascular plants, foliage is the primary receptor of photochemical pollutants. For photochemical oxidants to produce an effect in plants, they must come into contact with the leaf, pass through the stomata, and dissolve in the aqueous layer coating the cell walls.[274,283] Since stomata are the principal entry sites for ozone and PAN into plant leaves, stomatal closure presents a physical barrier to the entrance of oxidants and effectively protects the plant from injury.[185] Several studies suggest that oxidants may cause stomatal closure.[144,160,180,249] In one study, stomatal closure was associated with a genetic trait in onion wherein the stomata of sensitive plants did not close in response to ozone.[78] Dean[65] related the differences in ozone sensitivity between two tobacco cultivars to differences in stomatal density. Evans and Ting[82] found that maximum sensitivity of bean primary leaves was not associated with differences in stomatal number or leaf resistance to gas transfer. The effect of ozone and PAN on stomatal opening depends on both environmental and genetic factors.

Ozone can modify amino acids, proteins, unsaturated fatty acids, and sulfhydryl residues[113] located in cellular membranes. The initial effect of oxidants is to increase the leakage of water and ions, such as K^+ (potassium ion), from cells. In addition, ozone can induce the production of stress-induced ethylene.[1,50,283,294] The amount of ethylene produced in response to ozone stress is proportional to ozone concentration or duration of

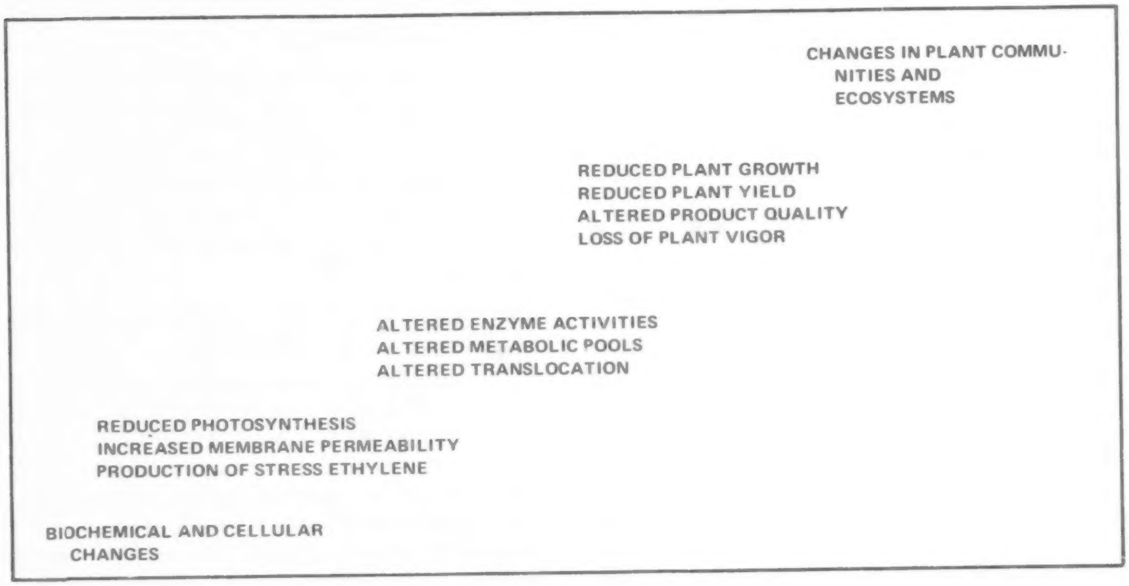

CHANGES IN PLANT COMMUNITIES AND ECOSYSTEMS
REDUCED PLANT GROWTH
REDUCED PLANT YIELD
ALTERED PRODUCT QUALITY
LOSS OF PLANT VIGOR
ALTERED ENZYME ACTIVITIES
ALTERED METABOLIC POOLS
ALTERED TRANSLOCATION
REDUCED PHOTOSYNTHESIS
INCREASED MEMBRANE PERMEABILITY
PRODUCTION OF STRESS ETHYLENE
BIOCHEMICAL AND CELLULAR CHANGES

Figure 11-1. Sequence of ozone-induced responses.[282]

exposure.[294] Plants sensitive to ozone produce more stress ethylene than less sensitive plants. Ethylene production is induced before the appearance of visible injury and frequently occurs without visible injury.

Todd[296] and Todd and Probst[298] also measured the effects of ozone at 7840 $\mu g/m^3$ (4 ppm) for 40 min on photosynthesis and found that the development of symptoms was associated with an inhibition of carbon dioxide fixation. This effect was also confirmed by Macdowall,[177] who reported that the inhibition of photosynthesis was greater than that which could be accounted for by chlorophyll destruction alone. Hill and Littlefield[130] associated decreased net photosynthesis caused by ozone (at 0.60 ppm for 1 hr) with both decreased stomatal opening and decreased transpiration. These studies have generally shown that net photosynthesis can be reduced without the association of visible injury.

The photosynthetic rate is an important indicator of plant vigor. In ponderosa pine, a reduction in this rate may occur at a low dose of ozone without causing visible symptoms. Miller et al.[210] found that a daily 9-hr exposure to ozone at 294 $\mu g/m^3$ (0.15 ppm) reduced apparent photosynthetic rates by 10 percent after 30 days, without typical ozone symptoms. Botkin et al.[23] found that a threshold ozone dose for suppression of net photosynthesis in eastern white pine was a 4-hr exposure to 980 $\mu g/m^3$ (0.50 ppm).

Ozone, in addition to inhibiting photosynthesis, alters the way in which the products of photosynthesis are distributed within plants. Results of studies in which alfalfa and ponderosa pine were exposed to low levels of ozone for a growing season indicated that sugars were retained in the foliage and were not translocated to the roots.[218,295] The resulting reduction of carbohydrates in the roots could reduce nitrogen fixation and plant growth.[93,260]

The vital physiological process of nitrogen fixation may be a key factor in the impact of photochemical oxidants on the plant community/ecosystem complex. Ozone is known to suppress nodulation in the roots of soybean[18,246,284] and ladino clover.[2,156] Blum and Tingey[18] found no direct effect of ozone on *Rhizobium* or on nodule formation. They attributed the decrease in nodule number and nitrogen fixation to the reduction in the available energy in the root tissues. Studies of the chronic exposure (7 hr per day) of alfalfa to 98 $\mu g/m^3$ (0.05 ppm) O_3 by Tingey[283] and Neely et al.[218] show that the total amount of fixed nitrogen was depressed by 40 percent. Weber[307] found that the effect of ozone and a mixture of ozone and sulfur dioxide on soybean resulted in suppression of nitrogen fixation by 50 percent. Suppression of atmospheric nitrogen fixation by root nodules could affect total biomass and agricultural yield, especially in areas of high oxidant pollution and low soil nitrogen. Kochhar[156] also reported an inhibition of plant growth and nodulation of *Trifolium repens* (clover) when plants were treated with root exudates taken from fescue grass that had been exposed to ozone. If these responses are of widespread occurrence, the competitive ability of plant species could be modified with a resulting change in plant diversity and a possible decrease in productivity.

The acute responses of plants to ozone and PAN result from disruption of normal cell structure and processes. The initial response results in cellular disorganization. Water and salts are lost from the cell interior; then plasmolysis results and cell death normally occurs. However, depending on dose and environmental conditions, membrane permeability may be restored and cell recovery occurs. The extent of recovery depends on the severity of the external stresses and on the ability of the cells to repair themselves. As indicated previously, the biochemical and physiological effects of ozone on plants are better understood than those of PAN.

Visible Symptoms

Visible symptoms are commonly used in characterizing the response of vegetation to a variety of stresses, including air pollution.[301] Different types of stress induce similar symptoms, thus causing difficulty in diagnosis. The visible symptoms resulting from oxidant injury to plants can be classified as belonging to two general categories—acute and chronic injury. Acute injury is usually manifested by cell destruction. Necrotic symptoms of acute injury are sometimes characteristic of a given oxidant. These patterns, at the least, demonstrate the effect of a chemical toxicant. Acute injury usually appears within 24 hr after exposure and is associated with short exposures (hours) to a specific oxidant or pollutant mixtures at relatively high concentrations. Chronic injury, whether mild or severe, is usually associated with long-term or multiple exposures to low concentrations of oxidants. Disruption of normal cellular activity occurs, leading to chlorosis or other pigment or color change. Cell death may eventually result. Chronic oxidant injury patterns may be confused with symptoms resulting from normal senescence and biotic pathogens, including insects, nutritional disorders, or other environmental stresses. These patterns may appear as premature leaf senescence.

Bobrov,[19,20,21,22] Bystrom et al.,[37] and Glater et al.[96] conducted the earliest studies of microscopic changes and developmental patterns resulting from simulated smog and ambient pollution. These workers first suggested that the initial effects were due to cell membrane injury. They described developmental patterns and showed that leaves near maximal expansion were most sensitive and that sensitivity was related to stomatal function, volume of intercellular spaces, and the extent of suberization of mesophyll cells. Other workers have since substantiated their studies.[192,276,281,286]

Although ozone and PAN are considered to be the two primary phytotoxic oxidants in the photochemical complex, the response of plants to simulated polluted atmospheric conditions suggests the existence of other ambient phytotoxic oxidants.[96] The symptoms associated with many of these reactant mixtures are closely related to those caused by ozone and PAN.[115,118] In some tests, however, the reactant mixtures used would not have produced either ozone or PAN. In other cases, the pattern of injury on sensitive test plants or age of tissue affected suggested the presence of one or more pollutants other than ozone or PAN. Plant injury symptoms observed in the field often resemble those reported from controlled exposures to ozone or PAN, but the response pattern in some cases is sufficiently different that accurate field diagnosis is difficult. Brennan et al.[30] correlated development of oxidant symptoms with aldehyde concentrations in New Jersey and suggested that aldehyde may be a major phytotoxic component of the photochemical oxidant complex. The symptoms observed were probably not in response to the aldehydes, but rather to a compound or group of compounds present under the same conditions as the aldehyde.[132] The concentration of the compound or group of compounds was probably directly related to the concentration of the aldehydes.

OZONE

The four general types of lesions found on plant leaves as a result of ozone injury are described in detail in *Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas.*[129] These are pigmentation (stippling), bleaching, foliar chlorosis, and bifacial necrosis. All except the last type of symptom occur primarily on the upper surface of the leaf.

1. *Pigmentation (stippling).* The palisade mesophyll cells of injured leaves take on brown, black, red, or purple colorations. The injury is evident primarily on the upper leaf surface. On many deciduous trees, shrubs, and some herbaceous plants, the accumulation of pigments in the dying cells results in small dot-like lesions.
2. *Bleaching (fleck).* Bleached or unpigmented lesions usually occur on upper leaf surfaces following collapse of the palisade cells and sometimes of the epidermal cells. Individual lesions are usually small and irregular in shape but may coalesce and become large, frequently resulting in slightly sunken areas on the upper leaf surface in certain herbaceous species.
3. *Foliar chlorosis.* Chlorotic areas usually occur on the upper surfaces of leaves, often coalescing to present a mottled appearance. Pine needles and grasses that do not possess differentiated mesophyll tissue may develop the chlorotic mottling on either leaf surface.
4. *Bifacial necrosis.* Bifacial necrosis results when the tissues connecting the upper and lower leaf surface are killed. The coloration of the tissue in the lesions may range from ivory to orange or brownish-red. Upper and lower surfaces often are drawn together, forming a thin, papery lesion.

The two classic symptoms (fleck and stippling) are more widely associated with the response of dicotyledonous plants to ozone than are other symptoms. Many plants (e.g., pinto bean, cucumber, tomato, soybean, and sycamore) may have the entire upper surface covered with a bleached appearance as a result of ozone exposure, with no observable injury on the lower surface. On closer examination, the bleached area is seen to be made up of many small groups of palisade cells that are dead and contain no pigment. In other plants or under different conditions, the palisade cells may accumulate dark alkaloid pigments (stipple) coincidentally with cell death. After exposure to higher ozone concentrations or after longer periods of exposure, injury extends to the spongy cells, producing bifacial necrosis. Plants exposed to a high concentration of ozone or to a high concentration of ambient pollution during a pollution episode usually develop dark, watersoaked areas in the leaf within a few hours. Leaves may show partial recovery, or these areas may form light-tan bifacial necrotic lesions within 24 to 48 hr. Individual lesions may be small, but groups of them can extend and affect a considerable portion of the leaf.

In monocotyledonous plants (grasses or cereals) and in some nonmonocotyledonous plants there is no division of mesophyll tissue, and injury usually appears as a bifacial fleck.[21]

The foregoing discussion is not descriptive of effects noted in coniferous trees such as white pine. Two classic oxidant (ozone) syndromes of pine, one in the eastern and the other in the western United States, have been described. Ozone is probably the cause of emergence tipburn in white pine (white pine needle dieback).[15] The injury is characterized as a tip dieback of newly elongating needles and occurs throughout the range of eastern white pine. Affected trees are found at random in a stand, and symptoms develop in discrete episodes in successive years.[217] Primary roots of affected trees often die after repeated needle injury. Costonis and Sinclair[48] reported silvery or chlorotic flecks, chlorotic mottling, and tip necrosis of needles as a result of ozone exposure.

Chlorotic decline, a needle injury of ponderosa pine, was first noticed in 1953 and was related to oxidant air pollution by 1961.[234] Chlorotic decline was characterized by a progressive reduction in terminal and diameter growth of the tree; only the current season's needles were retained. Yellow mottling and a reduction in the number and size of the remaining needles were noted. Eventually the tree died. The chlorotic decline was not associated with stresses other than ozone.[235]

PAN

The injury symptoms for the various peroxyacyl compounds are similar and cannot be distinguished. The characteristic symptoms are glazing, silvering, or bronzing of the lower surface of the leaves of plants such as spinach, garden beets, romaine lettuce, and chard. Symptoms on endive and turnip appear as a bleaching of the lower leaf surface that often develops into light tan necrotic areas. In some instances, upper leaf surfaces may also be affected.[216,217,238,272]

Microscopic examination of affected areas reveals a collapse of spongy mesophyll cells with a subsequent development of large intercellular air pockets, especially near the stomata. The air pockets give the leaves the glazed appearance.[212, 217,238]

Young expanding leaves are normally more sensitive to PAN than mature leaves. Leaves of plants such as grasses, tobacco, and petunia, which do not mature uniformly, may show leaf banding where injury is related to a sensitive region of the expanding leaf. Successive exposure to PAN may result in the formation of multiple bands.[20,238]

PAN symptoms are slow to develop and may take up to 72 hr for full development. A complete description of PAN injury to plants was written by Taylor and MacLean.[272]

Growth and Yield

Photochemical oxidants, including ozone and PAN, suppress growth and yield in many plant species. Although growth and yield effects are normally associated with visible injury, oxidants can inhibit growth with little or no injury. The basis for this variability is not understood, but probably it is associated with the genetic composition of plants, environmental condition, and pollutant combination and dose.

Attempts have been made to associate visible injury with other indices of plant response. Todd and Arnold[297] compared visible injury with biomass production and chlorophyll content of pinto bean exposed to a synthetic oxidant (ozone plus hexene). They noted a logarithmic relationship between injury and the two response parameters and suggested that leaf injury was not a reliable index for estimating growth losses. A group of Canadian workers[3,7] used physiological measurements exclusively to monitor the effects of acute ozone (oxidant) exposures. Because of the subjective nature of injury measurements and the often apparent poor correlation with growth responses, growth or physiological parameters are often evaluated directly.

AMBIENT AIR STUDIES

Since the first observations of oxidant injury by Middleton et al.,[203] there has been a continuing flow of reports mentioning the effects of oxidants on vegetation. The majority of these reports associate ozone or oxidant injury symptoms with episodes of given levels of oxidant or ozone. Engle et al.[80] in Wisconsin, Laurence et al.[167] and Kohut et al.[159] in Minnesota, Gardner[91] in South Dakota, Weaver and Jackson[306] and Haas[101] in Ontario, Rich et al.[247] in Connecticut, Daines et al.[54] in New Jersey, Brasher et al.[25] in Delaware, Skelly et al.[259] in Virginia, Reinert et al.[243] in Ohio, Tingey and Hill[289] in Utah, and Oshima et al.,[232] Cobb and Stark,[43] and Miller and Millecan[209] in California all noted plant effects from ambient oxidant. Haas[101] found that the rate of growth during oxidant exposure influenced the symptom severity, that the stage of growth determined the dose required to produce injury effects, and that the physical conditions within a field could cause variable plant responses to oxidant.

Cobb and Stark[43] and Miller and Millecan[209] reported the severe effects of ambient oxidant on ponderosa pine in the San Bernardino Mountains. The results of quantitative studies are reported in Chapter 12. Skelly et al.[259] associated post-emergence tipburn of eastern white pine with high levels of ambient oxidant.

Greenhouses with filtered and nonfiltered air or field chamber studies have been used to determine the effects of ambient pollutants on growth and yield of selected crops. Reductions in yield and biomass were some of the observed effects noted. Summary data from selected studies are shown in Table 11-1. Thompson and Taylor[280] summarized several years of detailed field chamber studies, during which lemon and orange trees were exposed to many pollutant combinations. The inhibiting effects of the pollutant on fruit size and number and on total yields were noted in these studies. The results of these studies provided the basis for the projected 50-percent reductions in citrus yields from the Los Angeles Basin as a result of photochemical oxidants. Thompson and Katz[279] showed that some of the effects noted on the citrus trees were probably related to concentrations of PAN in the ambient air. Thompson and coworkers,[277,278] using charcoal-filtered and unfiltered chambers to determine effects of ambient oxidants on grapes, found nominal yield effects in the first year and 50 to 60 percent reduction during the next 2 years. The year to year differences were attributed to the effects of oxidants on floral initiation the year before the first year study. A similar response may be expected for plants that initiate floral structures in the season previous to experimental treatments. A wax emulsion spray on the leaves gave a 20-percent increase in yield over unsprayed plants, suggesting that selected protectants may be useful in protecting grapes grown in areas of high oxidant concentration.

TABLE 11-1. EFFECTS OF OXIDANTS (OZONE) IN AMBIENT AIR ON GROWTH, YIELD, AND FOLIAR INJURY IN SELECTED PLANTS[a]

Plant species	Oxidant concentration, ppm	Duration of exposure	Plant response (reduction from control listed as %)	Reference
Lemon	>0.10	Over growing season	32, yield 52, yield (leaf drop and other effects)	280
Orange	>0.10	148 hr/month avg. from March-Oct.; 254 hr/month avg. from July-Sept.	54,yield (other reductions found)	280
Grape, cultivar	≥0.25	Often over May-September growing season	12, yield (first year) 61, yield (second year) (increased sugar content) 47, yield (third year)	277 278 275
Corn, sweet	0.20-0.35	Hourly maximum for 3 to 4 days before injury	67, injury (10 cultivars, 5 unmarketable) 18, injury (13 cultivars) 1, injury (11 cultivars)	38
Bean, white	>0.08	9 hr	(Bronze color, necrotic stipple, premature abscission)	306
Tobacco, cultivar Bel W_3	0.02-0.03	6 to 8 hr	(Minimal injury)	119
Tobacco, cultivar Bel W_3	>0.05	Often over growing season	22 (fresh wt), top 27 (fresh wt), root	108
Cotton, cultivar Acula	Ambient	Over growing season	7-20, lint + seed (3 locations, 1972) 5-29, lint + seed (3 locations, 1973)	33
Potato, 4 cultivars[b]	≥0.05	326 to 533 hr (2 years)	34-50, yield (2 years for 2 cultivars) 20-26, yield (1 year for 2 cultivars)	122
Potato, cultivar Haig	0.15	3 consecutive days	95, injury (leaf area covered)	25

[a] Table taken from Ref. 217.

[b] Greenhouse studies.

Other experimental studies noted the effects of ambient air levels of oxidant on cotton, potato, and tobacco yields. In the San Joaquin Valley, California, a 5- to 29-percent reduction in lint and seed was noted over a 2-year period when cotton was grown in charcoal-filtered vs unfiltered field chambers.[33] Heggestad[122] reported the results of a 3-year study comparing the growth of four potato cultivars in greenhouses with charcoal-filtered and unfiltered ambient air. Yields of sensitive cultivars were suppressed as much as 50 percent by ambient oxidants. Heagle et al.,[108] using open-top field chambers, reported preliminary results indicating a reduction in leaf yields of a sensitive tobacco cultivar when plants grown in ambient air and nonfiltered chambers were compared with plants grown in chambers with charcoal filters. The results of the research discussed in this section indicate that oxidants in the ambient air decrease plant growth and yield. Injury symptoms resulting from oxidant exposure have been noted on a nationwide basis. The most severe effects are found in California in the San Bernardino Mountains and the Los Angeles Basin.

CONTROLLED CHAMBER STUDIES

To define the effects of oxidants on plant growth and yields more accurately, many workers have used controlled additions of ozone to determine the effects of acute or chronic exposures on a variety of growth parameters. Although the results of the effects of ambient oxidants on vegetation may not be directly comparable with the results obtained in the controlled exposure studies discussed in the following sections, reduced yields and similar oxidant symptoms are common results. Most of the results discussed are from exposures in greenhouses and controlled environmental chambers, but several are from studies using exposure chambers placed over field plantings.

Short-Term Exposures — Adedipe and associates[3] reported reduced biomass and floral production in four bedding plants and reduced biomass in two radish cultivars[7] from acute ozone exposures (Table 11-2). They reported no effects on marigold, celosia, impatiens, and salvia cultivars, even at the high concentration of 0.80 ppm for a 2-hr exposure. Research involving radish has included the effects of exposure temperatures on the growth response to ozone.[7] One cultivar reacted to ozone in the same way regardless of temperature variation, but the response of the second cultivar was influenced by temperature. Tingey et al.[285] exposed radish at 7, 14, or 21 days of age and all combinations of these ages to ozone at 785 $\mu g/m^3$ (0.40 ppm) for 1.5 hr. For a single exposure, the greatest effect on root growth was noted on the 14-day-old cultivar; for double exposures, the greatest root growth effect occurred with the 7- and 14-day-old cultivars. However, the greatest reduction of root growth occurred with triple exposures of 7-, 14-, and 21-day-old cultivars. The reductions in root growth from the multiple ozone exposures were equal to the additive effects of three single exposures. It was concluded that root growth reductions resulted from the preferential use of photosynthate for foliar growth. Inhibition in the rate of root growth remained during the second week after exposure. Tingey and Blum[284] reported that when soybean plants were exposed to 1468 $\mu g/m^3$ (0.75 ppm) of ozone for 1 hr, root growth was consistently reduced more than top growth and, in addition, there was a reduction in nodule weight and number. Evans[81] exposed the middle leaflet of the first trifoliate leaf of pinto bean to various ozone concentrations. The results indicated a differential growth response in various leaf positions. A reduction in leaflet expansion was noted after a 12-hr treatment with 98 $\mu g/m^3$ (0.05 ppm) ozone. The foregoing discussion represents only a few of the representative examples regarding the effects of acute ozone exposure on plant growth.

Long-Term Exposures — Experimental long-term exposures of various crops as well as ornamental and native plants to ozone have resulted in a reduction in growth and/or yield. Harward and Treshow[105] exposed 14 species representative of the aspen plant community to ambient air containing ozone concentrations of 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm) and to ozone at 290 and 588 $\mu g/m^3$ (0.15 and 0.30 ppm) for 3 hr/day, 5 days/week, and to charcoal-filtered air throughout the growing season. In all species, foliar injury was seen at the highest pollution concentration (Table 11-3). There was considerable variability in the responses, and only six species formed seed; however, in most cases, growth was reduced and most species were sensitive. Price and Treshow[239] found major biomass reductions in six grass and two tree species exposed for 4 hr/day to ozone at 290 to 647 $\mu g/m^3$ (0.15 to 0.33 ppm) over a growing season. They also found reduced reproduction and a loss of some reproductive components. These effects on growth and reproduction could result in subtle shifts in community composition after several years of ozone exposure. Taylor et al.[271] reported a 52-percent reduction in the fresh weight of avocado seedlings exposed to a synthetic smog (ozone plus hexene) for 280 hr. Tingey et al.[293] found that the growth of two soybean cultivars (Hood and Dare) was inhibited by intermittent exposure to ozone at 196 $\mu g/m^3$ (0.10 ppm) for 3 weeks. A decrease in both root and top growth occurred. Similar results[287] were noted with radish, except that growth was inhibited at 98 $\mu g/m^3$ (0.05 ppm). The reduced growth noted in the aforementioned studies occurred even though there were very few visible symptoms of plant injury.

A 30-percent reduction in wheat yield occurred when at anthesis, wheat was exposed to ozone at 392 $\mu g/m^3$ (0.2 ppm) 4 hr/day for 7 days.[156] Oshima et al.[231] reported a reduction in tomato yield at 686 $\mu g/m^3$ (0.35 ppm) over an exposure period of 97 hr. Significant injury occurred at both 392 and 686 $\mu g/m^3$ (0.20 and 0.35 ppm) ozone. Plants exposed to the low ozone concentration tolerated a considerable amount of defoliation and a significant decrease in biomass without a corresponding reduction in yield. A significant reduction in yield, however, was recorded from plants in the higher (686 $\mu g/m^3$, or 0.35 ppm) ozone exposure where a greater decrease in biomass was observed. This yield reduction was caused by a lower fruit set and subsequently fewer harvested fruit. Oshima suggested an injury tolerance threshold for tomato below which no reduction in yield would occur. In an eariler study, Oshima[229] reported a decrease in kernel weight of sweet corn exposed to ozone for 4 percent of the growing period at concentrations of 392 or 686 $\mu g/m^3$ (0.20 or 0.35 ppm). The reduced weight was

TABLE 11-2. EFFECTS OF ACUTE EXPOSURE ON GROWTH AND YIELD OF SELECTED PLANTS[a]

Plant species	Ozone concentration, ppm	Exposure time, hr	Plant response (reduction from control listed as %)[b]	Reference
Begonia, cultivar White Tausendschon	0.10	2	5, avg. of 3 growth responses: shoot wt, flower wt, flower no.	3
	0.20	2	10, avg. of same responses	
	0.40	2	19, avg. of same responses	
	0.80	2	38, avg. of same responses	
Petunia, cultivar Capri	0.10	2	9, avg. of same responses	3
	0.20	2	11, avg. of same responses	
	0.40	2	21, avg. of same responses	
	0.80	2	31, avg. of same responses	
Coleus, cultivar Scarlet Rainbow	0.10	2	2, avg. of same responses	3
	0.20	2	17, avg. of same responses	
	0.40	2	24, avg. of same responses	
	0.80	2	39, avg. of same responses	
Snapdragon, cultivar Floral Carpet, mixture	0.10	2	0, avg. of same responses	3
	0.20	2	6, avg. of same responses	
	0.40	2	8, avg. of same responses	
	0.80	2	16, avg. of same responses	
Radish, cultivar Cavalier, Cherry Belle	0.25	3	36, top dry wt (Cavalier)	7
			38, root dry wt (Cherry Belle)	
Radish	0.40	1.5(1)[c]	37, root dry wt	285
		1.5(2)[c]	63, root dry wt	
		1.5(3)[c]	75, root dry wt	
Cucumber, cultivar Ohio Mosaic	1.00	1	19, top dry wt (1% injury)	226
	1.00	4	37, top dry wt (18% injury)	
Potato, cultivar Norland	1.00	4	0, tuber dry wt (no injury)	226
	1.00	4(3)[c]	30, tuber dry wt (injury severe)	
Tomato, cultivar Fireball	0.50	1	15, plant dry wt (grown in moist soil)	153
	1.00	1	20, plant dry wt (grown in moist soil)	
Tomato, cultivar Fireball	0.50	1	15, increase in plant dry wt (grown in dry soil)	153
	1.00	1	25, increase in plant dry wt (grown in dry soil)	
Onion, cultivar Spartan Era	0.20	24	0, effect	226
	1.00	1	19, plant dry wt (no injury)	
	1.00	4	49, plant dry wt	
Tobacco, cultivar Bel W_3	0.30	2	48, chlorophyll content	4

[a] Taken from Ref. 217

[b] Unless otherwise noted

[c] Number of exposures in parentheses

associated with a shriveled-ear condition (kernels) that might be related to the effects of ozone on pollen development.

Craker[49] reported a reduction in the weight of petunia flowers after plants were exposed to ozone for 53 days at 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm); however, an increase in the weight of petunia flower[52] was found when exposure to three different concentrations of ozone was for 7 days. Carnations (80 plants) continuously exposed for 38 days to 98 to 196 $\mu g/m^3$ (0.05 to 0.10 ppm) produced a single deformed flower, while the controls had 24 normal flowers.[84] Poinsettia bract area was decreased by 39 percent after a 50-day exposure (6 hr/day) to ozone at 196 to 235 $\mu g/m^3$ (0.10 to 0.12 ppm).[52]

Heagle and associates[110] found a reduction in yield of sweet corn and soybean[109] after exposure to ozone at 196 $\mu g/m^3$ (0.10 ppm) for 6 hr/day administered over much of the growing season. These exposures were carried out in field chambers set up over soybean and corn plots in the field. They suggest that a threshold for measurable effects on these crops would lie between ozone (oxidant) concentrations of 98 and 196 $\mu g/m^3$ (0.05 and 0.10 ppm) for 6 hr/day if exposures were continued throughout most of the growing season. These concentration values resemble growing-season averages found in the eastern United States.

In one of the few examples of chronic ozone studies involving forest tree species, Wilhour and Neely[310] exposed eight species of conifers to 200 $\mu g/m^3$ (0.10 ppm) of O_3 for 6 hr/day, 7 days/week, for 18 continuous weeks. Significant growth reduction (given as percent reduction from control) was observed in root length (12 percent), dry weight (DW) of stem (21 percent), and DW of root (26 percent) of ponderosa pine (*Pinus ponderosa* Laws), and in western white pine (*P. monticola* Dougl.) in DW of foliage (13 percent) and DW of stem (9 percent). Biweekly harvests of ponderosa pine (exposed as above) throughout a 20-week study period showed highly significant growth reductions in root DW (26 percent). A trend of increased growth differences associated with increased length of exposure to ozone was evident.

Bennett et al.[12] found experimental evidence for stimulation of growth at low concentrations of ozone. They exposed bean (cultivar Pure Gold Wax), barley (cultivar Brock), and smartweed to ozone at 59 $\mu g/m^3$ (0.03 ppm) and found instances of significant growth increases. The concept needs further study in light of the current concern over "normal" background ozone concentrations.

A number of significant investigations have been conducted that demonstrate the response of vegetation to long-term exposure of relatively low ozone concentrations. The results of these chronic ozone studies are summarized in Table 11-4. The results of such experiments suggest that overall effects on agriculture production could be extensive, depending on the sensitivity of the cultivars used.

To illustrate the relationship between ozone concentration, duration of exposure, and effects on foliage, growth, or yield, the data from Table 11-4 have been graphed (Figure 11-2). Each point on the graph represents a combination of concentration and time at which a significant reduction in growth, yield, or photosynthesis occurred. The dashed line is an indicator of the level below which

TABLE 11-3. EFFECTS OF OZONE ON SELECTED UNDERSTORY SPECIES FROM AN ASPEN COMMUNITY[a]

Plant species	Plant response at different ozone concentrations[b]								
	Foliar injury, % of control			Plant wt, % of control			Seed wt, % of control		
	Ambient air[d]	290 $\mu g/m^3$ (0.15 ppm)	588 $\mu g/m^3$ (0.30 ppm)	Ambient air[d]	290 $\mu g/m^3$ (0.15 ppm)	588 $\mu g/m^3$ (0.30 ppm)	Ambient air[d]	290 $\mu g/m^3$ (0.15 ppm)	588 $\mu g/m^3$ (0.30 ppm)
Chenopodium album L.	0	35	40	103	71[c]	83	87	99	87
C. fremontii L.	10	35	90	71	98	32	102	96	94
Descurainia sp.	0	15	55	179[c]	77	56[c]			
Geranium fremontii, L. Torr. ex A. Gray	7	50	90	93	94	53[c]			
Lepidium virginicum L.	50	100	100	121	72	31[c]	94	69	47[c]
Madia glomerata	40	100	100	112	38[c]	13[c]	79	74	18
Polygonum aviculare L.	30	95	95	271	186[c]	29[c]	127	43	50
P. douglasii Greene	5	35	95	87	29[c]	5[c]	93	84	3[c]

[a] Table from Ref. 217.

[b] Exposures were 3 hr/day, 5 days/week through growing season.

[c] Significant effect.

[d] Ambient air = 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm).

TABLE 11-4. EFFECTS OF LONG-TERM, CONTROLLED OZONE EXPOSURES ON GROWTH, YIELD, AND FOLIAR INJURY TO SELECTED PLANTS[a]

Plant species	Fig. 11-2 Nos.	Ozone concentration, μg/m³ (ppm)	Exposure time, hr	Plant response, percent reduction from control	Reference
Lemna, duckweed	1	196 (0.10)	5/day, 14 days	100, flowering; 36, flowering (1 wk after exposure completed) 50, frond doubling rate	86
Carnation	2	98-177 (0.05-0.09)	24/day, 90 days	50, flowering (reduced vegetative growth)	83
Geranium	3	137-196 (0.07-0.10)	9.5/day, 90 days	50, flowering (shorter flower lasting time, reduced vegetative growth)	83
Petunia	4	98-137 (0.05-0.07)	24/day, 53 days	30, flower fresh wt	49
Poinsettia	5	196-235 (0.10-0.12)	6/day, 5 days/week, 10 weeks	39, bract size	52
Radish	6	98 (0.05)	8/day, 5 days/week, 5 weeks	54, root fresh wt 20, leaf fresh wt	287
		98 (0.05)	8/day, 5 days/week (mixture of O_3 and SO_2 for same periods)	63, root fresh wt 22, leaf fresh wt	
Beet, garden	7	392 (0.20)	3/day, 38 days	50, top dry wt	223
Bean, cultivar Pinto	8	255 (0.13)	8/day, 28 days	79, top fresh wt 73, root fresh wt 70, height	182
Bean, cultivar Pinto	9	290 (0.15)	2/day, 63 days	33, plant dry wt; 46, pod fresh wt	139
	10	490 (0.25)	2/day, 63 days	95, plant dry wt; 99, pod fresh wt	
	11	686 (0.35)	2/day, 63 days	97, plant dry wt; 100, pod fresh wt	
Bean, cultivar Pinto	12	290 (0.15)	2/day, 14 days	8, leaf dry wt	175
	13	290 (0.15)	3/day, 14 days	8, leaf dry wt	
	14	290 (0.15)	4/day, 14 days	23, leaf dry wt (Data available on whole plants, roots, leaves, injury, and 3 levels of soil moisture stress)	
	15	290 (0.15)	6/day, 14 days	49, leaf dry wt	
	16	440 (0.225)	2/day, 14 days	44, leaf dry wt	
Bean, cultivar Pinto	17	440 (0.225)	4/day, 14 days	68, leaf dry wt (Data available on whole plants, roots, leaves, injury, and 3 levels of soil moisture stress)	175
	18	588 (0.30)	1/day, 14 days	40, leaf dry wt	
	19	588 (0.30)	3/day, 14 days	76, leaf dry wt	
Tomato	20	392 (0.20)	2.5/day, 3 days/week 14 weeks	1, yield; 32, top dry wt; 11, root dry wt	231
	21	686 (0.35)	2.5/day, 3 days/week, 14 weeks	45, yield; 72, top dry wt; 59, root dry wt	
Corn, sweet, cultivar Golden Jubilee	22	392 (0.20)	3/day, 3 days/week till harvest	13, kernel dry wt; 20, top dry wt; 24, root dry wt	229
	23	686 (0.35)	3/day, 3 days/week till harvest	20, kernel dry wt; 48, top dry wt; 54, root dry wt	
Wheat, cultivar Arthur 71	24	392 (0.20)	4/day, 7 days (anthesis)	30, yield	156
Soybean	25	98 (0.05)	8/day, 5 days/week 3 weeks	13, foliar injury	293
			8/day, 5 days/week (mixture of O_3 and SO_2 for same periods)	16, foliar injury 20, root dry wt	
Soybean	26	196 (0.10)	8/day, 5 days/week 3 weeks	21, top dry wt 9, root dry wt	
Alfalfa	27	196 (0.10)	2/day, 21 days	16, top dry wt	138
	28	290 (0.15)	2/day, 21 days	26, top dry wt	
	29	390 (0.20)	2/day, 21 days	39, top dry wt	
Grass, brome	30	290-647 (0.15-0.33) (varied)	4/day, 5 days/week growing season	83, biomass	239
Alfalfa[c]	31	196 (0.10)	6/day, 70 days	4, top dry wt, harvest 1 20, top dry wt, harvest 2 50, top dry wt, harvest 3	218
Alfalfa[c]	32	98 (0.05)	7/day, 68 days	30, top dry wt, harvest 1 50, top dry wt, harvest 2	218

TABLE 11-4. EFFECTS OF LONG-TERM, CONTROLLED OZONE EXPOSURES ON GROWTH, YIELD, AND FOLIAR INJURY TO SELECTED PLANTS[a] (cont'd).

Plant species	Fig. 11-2 No.[b]	Ozone concentration, $\mu g/m^3$ (ppm)	Exposure time, hr	Plant response, percent reduction from control	Reference
Alfalfa	33	98 (0.05)	8/day, 5 days/week 12 weeks	18, top dry wt	290
Pine, eastern white	34	196 (0.10)	4/day, 5 days/week 4 weeks (mixture of O_3 and SO_2 for same periods)	3, needle mottle (over 2-3 days of exposure) 16, needle mottle	69
Pine, ponderosa	35	290 (0.15)	9/day, 10 days	4, photosynthesis	210
	36	290 (0.15)	9/day, 20 days	25, photosynthesis	
Pine, ponderosa	37	290 (0.15)	9/day, 30 days	25, photosynthesis	210
	38	290 (0.15)	9/day, 60 days	34, photosynthesis	
	39	588 (0.30)	9/day, 10 days	12, photosynthesis	
	40	588 (0.30)	9/day, 20 days	50, photosynthesis	
	41	588 (0.30)	9/day, 30 days	72, photosynthesis	
	42	880 (0.45)	9/day, 30 days	85, photosynthesis	
Poplar, yellow	43	588 (0.30)	8/day, 5 days/week 13 weeks	82, leaf drop; O, height	147
Maple, silver	44	588 (0.30)	8/day, 5 days/week, 13 weeks	50, leaf drop; 78, height	147
Ash, white	45	588 (0.30)	8/day, 5 days/week 13 weeks	66, leaf drop; O, height	147
Sycamore	46	588 (0.30)	8/day, 5 days/week 13 weeks	O, leaf drop; 22, height	147
Maple, sugar	47	588 (0.30)	8/day, 5 days/week 13 weeks	28, leaf drop; 64, height	147
Corn, sweet, cultivar Golden Midget[c]	48	98 (0.05)	6/day, 64 days	9, kernel dry wt; 14, injury (12, avg. 4 yield responses)	110
	49	196 (0.10)	6/day, 64 days	45, 25, 35 for same responses	
Pine, ponderosa[c]	50	196 (0.10)	6/day, 126 days	12, root length 21, stem dry wt; 26, root dry wt	310
Pine, western white[c]	51	196 (0.10)	6/day, 126 days	13, foliage dry wt 9, stem dry wt.	310
Soybean, cultivar Dare[c]	52	98 (0.05)	6/day, 133 days	3, seed yield; 22, plant fresh wt; 19, injury, defoliation, no reduction in growth or yield	109
	53	196 (0.10)	6/day, 133 days	55, 65, 37 for same responses	
Poplar, hybrid	54	290 (0.15)	8/day, 5 days/week 6 weeks	50, shoot dry wt; 56, leaf dry wt; 47, root dry wt	148

[a] Modified from Ref. 217

[b] Numbers in this column are keyed to numbers in Fig. 11-2.

[c] Studies conducted under field conditions, except that plants were enclosed to ensure controlled pollutant doses. Plants grown under conditions making them more sensitive.

significant effects probably do not occur. In general, as the length of exposure increases up to 15 days, the average ozone concentration must be decreased to prevent growth effects. Beyond 15 days, if the average concentration of ozone exceeds 98 $\mu g/m^3$ (0.05 ppm), significant growth and yield effects could occur.

Quality — A limited number of studies have investigated the effects of ozone on crop quality. Thompson and his co-workers[277,278] compared the quality of grape juice made from grapes harvested from plants grown for two seasons in either ambient air oxidants or in carbon-filtered air. Sugar content of the grapes grown in ambient air was reduced 13 to 17 percent, but the concentration of organic acids showed no detectable trends. Frey[89] reported a 5-percent decrease in lipids and a 21-percent increase in amino acids in seeds from soybeans exposed to ozone. Exposure of alfalfa to 98 $\mu g/m^3$ (0.05 ppm) or to 196 $\mu g/m^3$ (0.10 ppm) ozone increased the levels of protein and amino acids, had no effect on carbohydrates or lignin, and decreased β carotene per unit weight of tissue.[218] The protein increase per unit weight of tissue is of no practical significance, because the ozone exposure produced a reduction in growth that resulted in a decrease in total production of protein.

DOSE-RESPONSE RELATIONSHIPS

An understanding of dose-response relationships is important for a basic understanding of the mechanism of oxidant effects on plants. Ideally this relationship could be expressed by a set of standard equations that would relate plant response to pollutant concentration and duration of exposure and would also incorporate the effects of all other factors that control the responses of plants. Development of such equations requires a data base sufficient to relate a given dose (concentration of pollutant times duration of exposure) of oxidant (e.g., ozone or PAN) to some meaningful plant effect. Such equations are not yet available; however, discussions of the relationship of time, concentration, and response have been published.[117,174]

Limiting Values — The concept of limiting values may be used to define the boundary between doses of a pollutant that are likely to cause measurable injury to an organism and those that are not likely to cause injury.[145] Jacobson[145] used this concept to estimate limiting values for the exposure of trees, shrubs, and agricultural crops to ozone and PAN. In estimating limiting values, several factors were taken into consideration. These were: (1) Of the known effects on vegetation, which were the most important? (2) What kinds of data should be included—data from the field, where ambient pollutant concentrations were monitored, or from the results of experimental fumigations with controlled dosages of the pollutant? (3) Which model should be used to estimate limiting values? Alteration of biochemical or physiological processes, reductions in growth or yield, or foliar lesions are effects on vegetation that can be considered. However, most of the published data discussing dose-response relationships use visible foliar injury as the index of response. Consequently, Jacobson[145] in estimating limiting values, used the dose-response data for visible foliar injury on the assumption that prevention of foliar injury would also prevent other adverse effects. In addition, in deriving limiting values, only data from experimental investigations in which both concentration and duration of exposures were monitored or controlled were used. More than 200 studies were surveyed to obtain the data. The concentration-time model developed by Jacobson for summarizing the dose-response relationships for effects of oxidants on vegetation

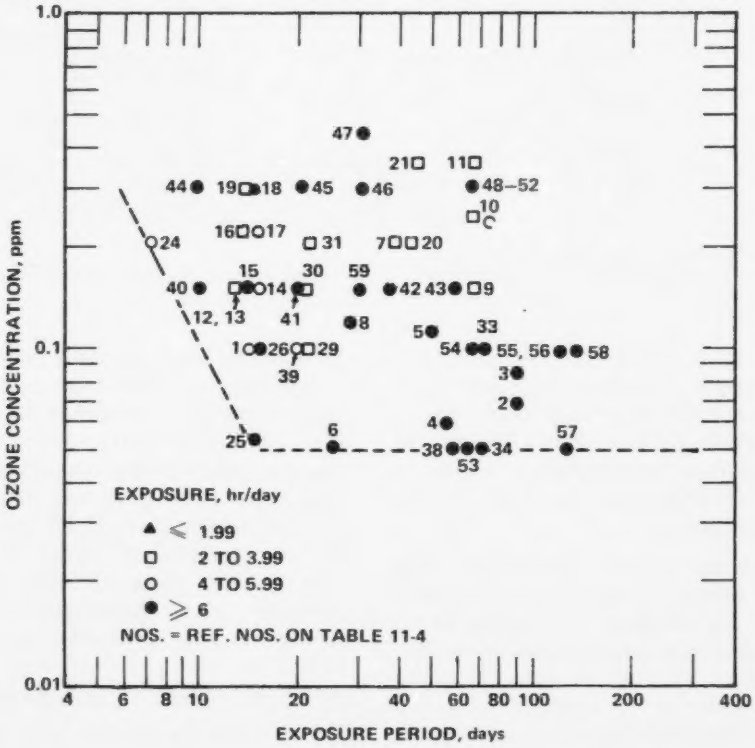


Figure 11-2. Relation between ozone concentration, foliar injury, and a reduction in plant growth or yield (see Table 11-4).

is similar to the one used by McCune[189] for fluorides and by Larsen and Heck[166] for ozone and SO_2. In the model, foliar injury is expressed as a function of the concentration of ozone or PAN and the logarithm of the duration of exposure.

Limiting Values for the Effects of Ozone — A single coordinate point for each published study was used in developing the limiting values in Figures 11-3 and 11-4. The single point represents the lowest concentration and duration of exposure in the published study that resulted in foliar symptoms. The data points used to develop the limiting values that will protect vegetation against oxidant-induced foliar injury are not necessarily threshold values. Since these points are based on available published research data (more than 100 studies), the shortcomings that were present in the experiments will be reflected in the derived points (e.g., shortcomings in calibration and measurement techniques and the use of subjective methods to determine foliar injury).

Woody Trees and Shrubs. The limiting values for the effects of ozone on trees and shrubs (Figure 11-3) are based on a single coordinate corresponding to the lowest concentration of ozone and duration of exposure that produced foliar injury on woody plants. The data used to establish the coordinates came from 18 studies.[13,14,17,47,56,57,60,126,131,152,158,161,168,171,211,250,253,311] The shaded area in Figure 11-3 depicts the range of uncertainty and refers to the range, as given in the reports, of the lowest doses of ozone observed to cause foliar injury. Doses of ozone above and to the right of the shaded area are likely to cause foliar injury to susceptible woody plant species. There is little likelihood that doses of ozone below and to the left of the shaded area will cause foliar injury.

Agricultural Crops. More data (over 100 studies) on dose-response relationships for agricultural crops are available than for any other class of vegetation. A wide range of uncertainty exists for the limiting values as given in Figure 11-4 because of the variability in plant response. In addition, the limiting values (shaded area, Figure 11-4) overlap the range of ozone concentrations producing 5 percent injury to crops as reported by Heck and Tingey[121] (Figure 11-5, Tables 11-5 and 11-6), and the response threshold for ozone effects on plants, published by the National Research Council of Canada,[238] corresponds closely to the lower curve in Figure 11-4. Doses of ozone above and to the right of the shaded area in Figure 11-4 will probably cause foliar injury in susceptible agricultural

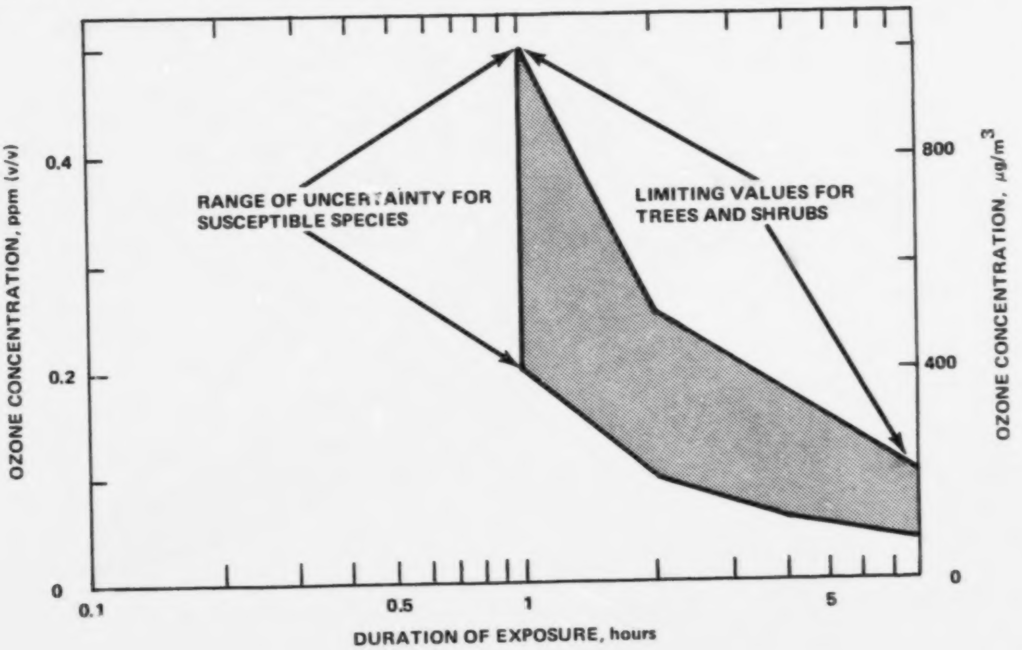


Figure 11-3. Limiting values for foliar injury to trees and shrubs by ozone.[145]

crops. Doses below and to the left of the shaded area are unlikely to cause foliar injury. The dose-response data used in establishing the limiting values in Figure 11-4 were obtained from many sources[98,117,174,238] (see also author citations, Table 11-2). The data available at present are not sufficient to determine if the curves in Figure 11-4 become parallel to the horizontal axis or, if at durations of 8 hr or more, they approach the axis asymptotically. The inaccuracies in measurements make the interpretation of results of repeated or long-duration exposures difficult, and limiting values of ozone concentrations below 100 $\mu g/m^3$ (0.05 ppm) are not useful.[145]

Limiting Values for the Effects of PAN — The limiting values for PAN are represented by the solid line in Figure 11-6. Appreciable risk of foliar injury to susceptible species of vegetation will occur above and to the right of the limiting values. A low risk of foliar injury exists below and to the left of the limiting values. The limited information base for the effects of PAN makes it impossible to estimate a range of uncertainty at present.

CONCLUSIONS

Based on the data available, the limiting values for the effects of ozone on trees and shrubs range from 400 to 1000 $\mu g/m^3$ (0.2 to 0.5 ppm) for a duration of 1 hr, 200 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 2 hr, and 120 to 340 $\mu g/m^3$ (0.06 to 0.153 ppm) for 4 hr. For agricultural crops, the ranges for limiting values are 400 to 800 $\mu g/m^3$ (0.2 to 0.41 ppm) for 0.5 hr, 200 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 1 hr, and 75 to 180 $\mu g/m^3$ (0.04 to 0.09 ppm) for 4 hr.[145] These values are similar to those of Heck and Tingey[121,217] for sensitive vegetation. These were: 400 $\mu g/m^3$ (0.2 ppm) for 1 hr, 200 $\mu g/m^3$ (0.1 ppm) for 2 hr, and 98 $\mu g/m^3$ (0.05 ppm) for 4 hr. Limiting values for oxidant exposures longer than 4 hr are not significant because of inaccuracies in measuring low concentrations.

The limiting values for the effects of PAN on agricultural plants are: 1000 $\mu g/m^3$ (0.2 ppm) for 0.5 hr, 500 $\mu g/m^3$ (0.1 ppm) for 1 hr, and 175 $\mu g/m^3$ (0.035 ppm) for 4 hr. The absence of data distinguishing the effects of PAN on woody plants or agricultural crops makes the separation of the data impossible.

ECONOMIC ASSESSMENT

This discussion will bring into focus our present understanding of the economic effects of oxidant air pollution on vegetation. Heck and Brandt[117] presented in depth the problems inherent in

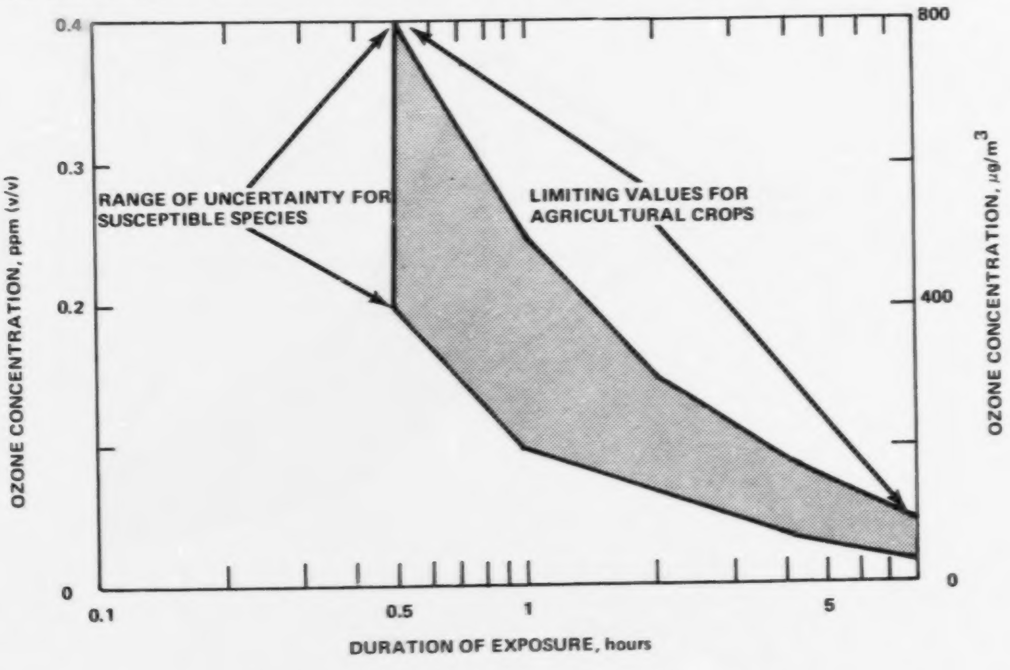


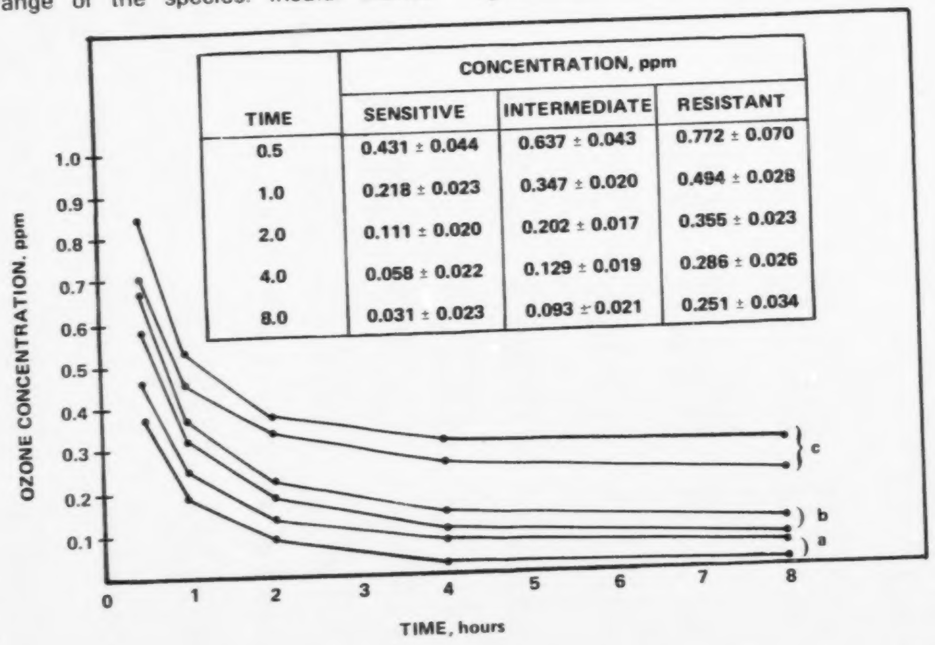
Figure 11-4. Limiting values for foliar injury to agricultural crops by ozone.[146]

making an economic evaluation of the response of plants to air pollutants, including ozone and other oxidants. They used the distinction between injury and damage proposed by German workers.[98] *Injury* is defined as any indentifiable and measurable response of a plant to air pollution; *damage* is any measurable adverse effect on the desired or intended use of the plant. Hence, before an effect on a plant can be evaluated in terms of economics, the plant must have been altered either quantitatively or qualitatively in such a way as to reduce its use value. In this context, visible symptoms or transient changes in physiologic responses may not result in an economic loss. For example, leaf necrosis in soybean is injury; to be classified as damage, the injury must affect bean yield. In contrast, an oxidant episode that bronzes the leaves of romaine lettuce may not affect biomass, but it will affect use and may result in complete economic loss. Similarly, ornamental plants, whose major use value depends on appearance, may be both injured and damaged.

Emergence tipburn of eastern white pine[15] and other physiogenic diseases of white pine are associated with air pollution throughout the natural range of the species. Insular stands containing ponderosa pine are frequently injured in the Southwest. The significance of the physiogenic diseases is not understood because only sensitive genotypes are affected. The disease occurs randomly in the forest, and its most obvious effect is the gradual elimination of genotypes that may have otherwise superior silvicultural characteristics. This could be a serious loss to future tree improvement efforts, and similar effects may be occurring in other forest species. Economic considerations have not been addressed in any reasonable way.

Oshima and his coworkers[227,228,230] have developed a methodology for evaluating and reporting economic crop losses that involves continuous air monitoring, chamber exposures, and monitoring plant species. Oshima has attempted to weld these parameters into a comprehensive method of determining yield reductions. Several species have been investigated, but the procedure has not been fully clarified. No economic analyses of the yield reductions were presented. This type of approach should be investigated further.

Any attempt to assess oxidant damage to agricultural crops requires judgement by a



TIME	CONCENTRATION, ppm		
	SENSITIVE	INTERMEDIATE	RESISTANT
0.5	0.431 ± 0.044	0.637 ± 0.043	0.772 ± 0.070
1.0	0.218 ± 0.023	0.347 ± 0.020	0.494 ± 0.028
2.0	0.111 ± 0.020	0.202 ± 0.017	0.355 ± 0.023
4.0	0.058 ± 0.022	0.129 ± 0.019	0.286 ± 0.026
8.0	0.031 ± 0.023	0.093 ± 0.021	0.251 ± 0.034

Figure 11-5. Ozone concentrations versus duration of exposure required to produce a 5% response in three different plant susceptibility groupings. The curves were generated by developing 95% confidence limits around the equations for "all plants" in each susceptibility grouping from Table 11-5. Curves: a=sensitive plants, b=intermediate plants, c=resistant plants.[217]

competent investigator. Landau and Brandt[165] suggest that the accuracy of crop surveys is directly related to the number of subjective decisions required in data collection. The first surveys were conducted in California[203,204] and were used, with a general survey of conditions on the east coast, to develop some of the early economic loss estimates of $8 to $10 million in California and $18 million on the east coast.[217] These estimates were raised to $500 million on the basis of increased awareness of pollution effects and increased recognition of additional sensitive species. However, all early estimates were subjective, with no substantial backup data.

Since 1969, a number of states have instituted an intensive training program for county agricultural agents and have made detailed reports of crop injury and damage[87,164,165,205,214,236] (Table 11-7). The first such report came from Pennsylvania[164] in 1969 and gave an estimate of a $9.6 million loss to agronomic commodities from oxidant pollutants. This survey included direct and some indirect costs. Similar surveys have been conducted in New England,[214] New Jersey,[236,237] and California.[205] These surveys considered yield reductions on the basis of injury and made no direct assessments of growth or yield, although subjective estimates of damage were obtained. Pell and Brennan[237] presented a well developed thesis on the rationale for differences in estimating losses to agriculture in New Jersey between 1971 and 1972. This subject was discussed in relation to the overall problem of assessing agricultural losses.

A study was initiated in 1969[11] by Stanford Research Institute (SRI) to develop an empirical model for assessing damage to vegetation. This program made use of laboratory and field data from controlled exposures of various crops and of chemical data from simulated reaction chambers so that estimates of ozone and other oxidants could be made on the basis of concentrations of primary pollutants. Hydrocarbon was chosen as the basic pollutant from which to develop the model for

TABLE 11-5. CONCENTRATION, TIME, AND RESPONSE EQUATIONS FOR THREE SUSCEPTIBILITY GROUPS AND FOR SELECTED PLANTS OR PLANT TYPES WITH RESPECT TO OZONE[a]

Plants	$(C = A_0 + A_1 I + A_2/T)$[b]	R^2[c]	Threshold concentration, ppm[d]			No. data points	Mean values[e]			
			1 hr	4 hr	8 hr		Conc. (C), ppm	Time (T), hr	Response (I), %	Dose, ppm/hr
Sensitive:										
All plants	-0.0152 + 0.0040I + 0.213/T	0.57	0.22	0.06	0.03	471	0.29	1.74	45.4	0.503
Grasses	-0.0565 + 0.0048I + 0.291/T	0.74	0.26	0.04	0.01	71	0.37	1.66	50.9	0.608
Legumes	0.0452 + 0.0036I + 0.172/T.	0.46	0.24	0.11	0.09	100	0.34	1.42	40.1	0.480
Tomato	-0.0823 + 0.0043I + 0.243/T	0.50	0.18	None	None	20	0.31	1.50	56.5	0.491
Oat	-0.0427 + 0.0051I + 0.273/T	0.76	0.26	0.05	0.02	30	0.37	1.66	40.2	0.611
Bean	-0.0090 + 0.0030I + 0.164/T	0.58	0.17	0.05	0.03	62	0.30	1.23	47.2	0.370
Tobacco	0.0245 + 0.0034I + 0.137/T	0.52	0.18	0.08	0.06	197	0.23	1.90	38.9	0.448
Intermediate:										
All plants	0.0244 + 0.0065I + 0.290/T	0.74	0.35	0.13	0.09	373	0.37	1.67	27.0	0.625
Vegetables	-0.0079 + 0.0064I + 0.263/T	0.79	0.29	0.09	0.06	25	0.41	1.29	33.5	0.532
Grasses	0.0107 + 0.0059I + 0.292/T	0.82	0.33	0.11	0.09	68	0.39	1.61	31.0	0.625
Legumes	0.0116 + 0.0074I + 0.329/T	0.81	0.38	0.13	0.09	104	0.40	1.59	25.0	0.642
Perennial	0.0748 + 0.0070I + 0.237/T	0.77	0.35	0.17	0.14	27	0.36	1.91	22.9	0.687
Clover	-0.0099 + 0.0071I + 0.268/T	0.95	0.29	0.09	0.06	24	0.28	2.13	23.0	0.595
Wheat	-0.0036 + 0.0081I + 0.302/T	0.88	0.34	0.11	0.08	15	0.47	1.25	28.9	0.508
Tobacco	0.0631 + 0.0087I + 0.152/T	0.78	0.26	0.14	0.13	59	0.28	1.99	15.7	0.551
Resistant:										
All plants	0.1689 + 0.0095I + 0.278/T	0.51	0.50	0.27	0.25	291	0.45	1.55	10.6	0.696
Legumes	0.0890 + 0.0108I + 0.304/T	0.82	0.45	0.22	0.18	36	0.30	1.89	12.2	0.722
Grasses	0.1906 + 0.0117I + 0.263/T	0.55	0.51	0.31	0.20	13	0.45	1.47	6.5	0.655
Vegetables	0.1979 + 0.0126I + 0.107/T	0.70	0.38	0.29	0.20	16	0.55	1.50	17.8	0.819
Woody plants	0.2312 + 0.0061I + 0.208/T	0.45	0.47	0.31	0.30	46	0.39	2.50	7.8	0.905
Cucumber	0.1505 + 0.0141I + 0.106/T	0.83	0.33	0.25	0.23	18	0.41	1.41	13.3	0.581
Chrysanthemum	0.2060 + 0.0052I + 0.256/T	0.40	0.49	0.30	0.27	45	0.39	2.17	12.6	0.847

[a] Equations were developed from exposures limited in time (0.5 to 8 hr, except for 2 to 12 hr points in the sensitive group) and denote acute responses of the plants. Concentrations range from 0.05 to 0.99 (1.0) ppm and responses from 0 to 99 (100)% of control. Reference 217.

[b] C is ozone concentration in ppm, I is percent injury, T is time in hours, and A_0, A_1, and A_2 are constants (partial regression coefficients) that are specific for pollutant, plant species or group of species, and environmental conditions used.

[c] Multiple correlation coefficient squared, which represents the percent variation explained by the model.

[d] For 5% response in 1-, 4-, and 8-hr periods.

[e] From the computer analysis.

prediction of expected oxidant values and, therefore, effects on various crops. From the oxidant value, injury and damage for specific crops were calculated for over 100 statistical reporting areas in the United States. The report used many subjective assumptions and was related primarily to visible injury symptoms. On the basis of the SRI model, the 1969 estimated loss to vegetation from oxidants in the United States was approximately $125 million. According to the National Research Council,[217] if the increase in crop values are considered and if it is assumed that oxidant concentrations have not been significantly reduced during the last few years, the loss in 1974 from oxidants could approach $300 million as based on the SRI report.

A summary of estimates derived from various surveys and assessment techniques is shown in Table 11-7. These values are suggestive at best. As with all values developed for agricultural losses, these have been directed at losses to the producer—not the consumer.

Whereas a reduction in production may actually increase the aggregate farm income and produce serious income distribution problems, the consequent reduction in marketable surplus would cause a significant rise in the cost to consumers because of the inelastic consumer demand for most agricultural crops. Thus at the consumer level, losses based on farm prices are not appropriate and are likely to be conservative. Because of percentage markups and fixed wholesale and retail marketing costs, the cost to the consumer of agricultural losses could be twice as great as that observed at the farm level.

TABLE 11-6. OZONE CONCENTRATIONS FOR SHORT-TERM EXPOSURES THAT PRODUCE 5 OR 20 PERCENT INJURY TO VEGETATION GROWN UNDER SENSITIVE CONDITIONS[a]

	Ozone concentrations that may produce 5% or 20% injury, ppm[b]		
Exposure time, hr	Sensitive plants	Intermediate plants	Resistant plants
0.5	0.35 - 0.50 (0.45 - 0.60)	0.55 - 0.70 (0.65 - 0.85)	≥0.70 (0.85)
1.0	0.15 - 0.25 (0.20 - 0.35)	0.25 - 0.40 (0.35 - 0.55)	≥0.40 (0.55)
2.0	0.09 - 0.15 (0.12 - 0.25)	0.15 - 0.25 (0.25 - 0.35)	≥0.30 (0.40)
4.0	0.04 - 0.09 (0.10 - 0.15)	0.10 - 0.15 (0.15 - 0.30)	≥0.25 (0.35)
8.0	0.02 - 0.04 (0.06 - 0.12)	0.07 - 0.12 (0.15 - 0.25)	≥0.20 (0.30)

[a] Data developed from analysis of acute responses shown in Table 11-5 and Figure 11-5. Reference 217

[b] 1 ppm = 1960 $\mu g/m^3$.

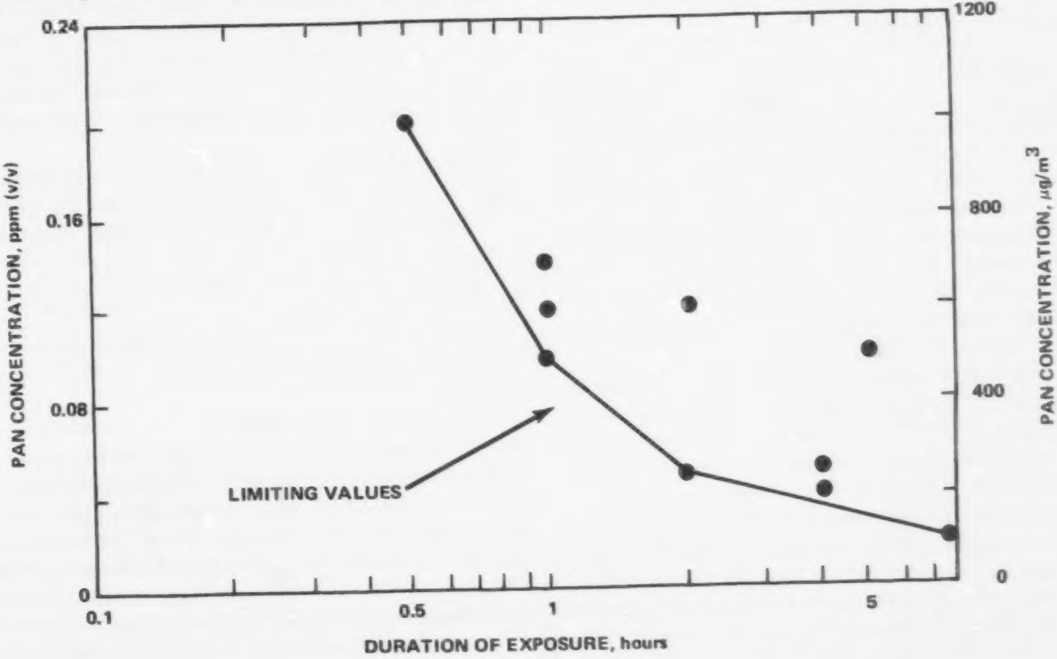


Figure 11-6. Dose-response relationships and limiting values for foliar injury to vegetation by peroxyacetylnitrate (PAN).[145]

TABLE 11-7. ESTIMATES OF ECONOMIC LOSSES TO CROPS AND VEGETATION IN THE UNITED STATES ATTRIBUTABLE TO OXIDANT AIR POLLUTION[a][217]

Area and year		Estimated loss, $\$10^3$	Comments	Reference
United States	1963	\$ 65,000	First approximation for commercial crops (SRI)	11
		121,400	Revised SRI report to include ornamentals	11
California	1963	33,700	Revised SRI report	11
	1970	17,500	Does not include ornamentals or indirect costs	205
Pennsylvania	1963	6,300	Revised SRI report	11
	1969	9,600	Pa. survey; includes indirect costs	164
	1970	60	Pa. survey, as above	163
New Jersey	1971	960	N. J. survey of a limited number of crops, based on visible injury	87
	1972	60	N. J. as above	236
New England	1971	1,100	Mass. survey; primarily crops and ornamentals	214

[a] Some of variation reflects methodology; other reflects differences in plant susceptibility and pollution over years.

Factors Affecting Plant Response

The response of plants to ozone, PAN, or any environmental stress is conditioned by complex, interacting internal and external factors. These include climatic and edaphic factors, interactions among atmospheric pollutants, genetic variability (both between and within species) of plants, the growth and physiologic age of susceptible plant tissue, and interaction of the plants with a variety of pathogens. A conceptualization of the interacting factors involved in air pollution effects on vegetation is shown in Figure 11-7.

GENETIC FACTORS

Resistance to ozone and PAN varies among species of a given genus and among cultivars within a given species. Variations in response are functions of genetic variability and environmental stresses as they affect morphological, physiological, and biochemical characteristics. In native populations and in breeding experiments, both ozone and PAN may act as stresses influencing selective pressure. Variations in species response to ozone and PAN are well documented.[126,129,150,168,270,302,312]

A number of papers discussing the variation in response of cultivars within species have been published.[123–125,250] The research of Brennan et al.[31] indicated that cultivars of oats and potato responded differently to ozone. Heck [114] summarized the variation in response of cultivars within several plant species, while Reinert[241] compiled a compendium of papers that discussed the response of cultivars of horticultural crops to ozone, PAN, and other pollutants. Variations in the responses of cultivars within three species have been studied intensively to determine their sensitivity to ozone. They are: petunia,[25,40,85] tomato,[41,42] and tobacco.[34,66,97,192–195]

Additional studies discussing cultivar responses to ozone are available for bean,[58] begonia,[3] morning glory,[215] chrysanthemum,[27,155] poinsettia,[183] spinach,[184] lettuce,[244] radish,[244] turfgrasses,[26] forage legumes,[32] alfalfa,[141] safflower,[143] soybean,[142,206,291] small grains (oat, rye, wheat, barley),[242] and English holly.[28] Petunia has been studied with regard to both auto exhaust and PAN,[85] and the effects of PAN alone on both petunia and chrysanthemum have been studied.[71,313] Most of these studies reported the results of acute exposures, which may or may not be related to results from chronic exposures. Foliar injury was generally used as a measure of response. It is not known whether rankings according to foliar injury relate to loss in economic yield.

Hanson[103] published a list of 160 woody species that were sensitive or tolerant to oxidants, based on observations made at the Los Angeles State and County Arboretum. A number of recent investigations have considered susceptibility of tree species to ozone.[14,17,47,55–57,60,126,147,206,310] A limited number of studies discuss selection of resistant individuals within native tree species.[14,60,210,253]

Two studies have explored the mechanism of genetic resistance to ozone. Engle[77] and Engle and Gableman[79] found that ozone sensitivity in onion is probably controlled by a single gene pair, with dominance of the resistant gene. In resistant plants, the membrane of the guard cells was more sensitive to ozone; when exposed to ozone, it lost its differential permeability, thus causing stomatal closure. This did not occur in the guard cells of the sensitive cultivar, and thus the stomata remained open. Taylor[267] crossed the sensitive Bel W_3 tobacco with a resistant line and found that the F_1 generation was of intermediate sensitivity. The F_2

generation segregated into 40 percent resistant, 10 percent sensitive, and 50 percent intermediate. He suggested that sensistivity was controlled by at least two genes. Resistance mechanisms to air pollutants are poorly understood, and further studies are needed.

A study using *Arabidopsis thaliana* was conducted to determine the potential mutagenic effects of ozone.[35] This plant completes its life cycle in about 35 days. Many generations may therefore be grown within a relatively short period of time. Plants were exposed to acute doses of ozone for 6 hr/day, 3 days/week, throughout the life cycle. Seeds were collected from control and exposed plants and planted for seven generations. Seed production and biomass were reduced within a specific generation, but none of the factors studied were transmitted to subsequent generations. The results showed that for this particular species, the concentrations of ozone used produced no mutagenic effects.

The aforementioned studies suggest that a spectrum of genotypes exists that varies in susceptibility to oxidants, and that environmental factors are important in conditioning a plant's susceptibility to pollutants. The degree to which environmental modification can control the response of sensitive genotypes to ozone may be less pronounced if specific biochemical or physiological mechanisms involved in plant response to pollutants have weak interactions with the environment.

PHYSIOLOGICAL AGE

The physiological age of a plant is an important factor in modifying plant sensitivity. Young, rapidly expanding leaves are most sensitive to PAN injury.[20,238] Ozone, in contrast, affects more

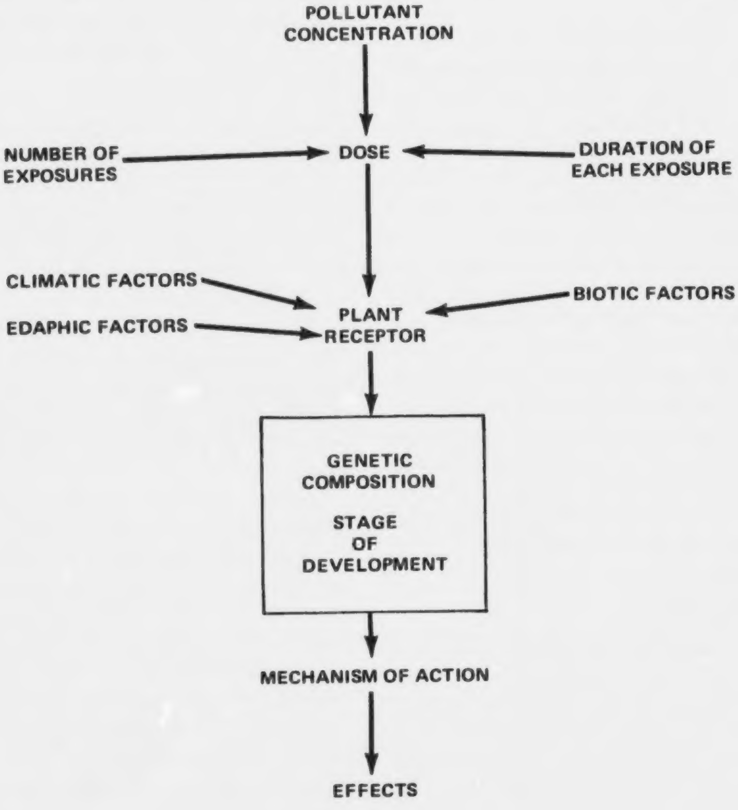

POLLUTANT CONCENTRATION
NUMBER OF EXPOSURES
DOSE
DURATION OF EACH EXPOSURE
CLIMATIC FACTORS
EDAPHIC FACTORS
PLANT RECEPTOR
BIOTIC FACTORS
GENETIC COMPOSITION
STAGE OF DEVELOPMENT
MECHANISM OF ACTION
EFFECTS

Figure 11-7. Conceptual model of factors involved in air pollution effects on vegetation (modified from van Haut and Stratmann).[302]

mature tissue. Maximum sensitivity is found when cotton leaves are approximately 70 percent expanded,[281] red maple 90 percent,[299] and soybean 60 to 80 percent.[286] In tobacco, maximum sensitivity occurs just after full leaf expansion.[176]

Davis[55] and Davis and Wood[60] found that the age of Virginia pine needles influenced their response to a 4-hr exposure to ozone at 490 $\mu g/m^3$ (0.25 ppm). Generally, cotyledons were more sensitive than primary needles, which in turn were more sensitive than secondary needles. Secondary needles of seedlings were approximately as sensitive as those of 3-year-old trees. The cotyledons and secondary needles became resistant after 16 and 18 weeks of age, respectively, whereas the primary needles remained sensitive beyond 18 weeks. Berry[13] reported that Virginia, shortleaf, loblolly, and slash pines at 2 to 6 weeks from seed were most sensitive when exposed to ozone at 490 $\mu g/m^3$ (0.25 ppm) for 2-hr.

Hanson et al.[104] reported increased tolerance in petunia cultivars to ozone as they approached the flowering stage. This was true for both ozone-sensitive and tolerant cultivars, with the latter being the more strongly influenced. They suggested that bud development resulted in the production of diffusible substances that moved down the plant and acted as a protectant.

Generally, studies have shown that plants are most sensitive to ozone at a physiologic age associated with full leaf expansion. Sensitivity is also associated with functional stomata, intercellular spaces, and rate of cutin formation on cell walls.[20] Plants generally are more sensitive to oxidants during stages of rapid growth and less sensitive as leaves mature. When oxidant episodes occur throughout the growing season, the older leaves, weakened during their stage of maximal physiologic growth, senesce prematurely.

CLIMATIC CONDITIONS

Plant response to ozone and PAN may be altered by climatic conditions before, during, and after exposure.[114] Plants may be sensitized by a given set of conditions after 1 to 5 days.

Conditions before and during exposure are critical, and those after exposure may be important. The responses of plants to ozone and PAN under varied climatic conditions were studied primarily under laboratory and greenhouse conditions. Field observations have often substantiated these results. Most studies have involved individual environmental factors and one or two response measures, including usually the evaluation of injury. Several investigations have dealt with the interactions of environmental factors. Information sufficient to make generalizations regarding plant response to oxidants exists, but there is much uncertainty because of the small number of species studied and the lack of information on the interactions of the environmental factors evaluated.

Light Quality — The quality of light affects the growth and development of plants and plays a role in determining the response of plants such as pinto bean to PAN.[72,73] Injury to pinto bean from PAN was maximal when exposed to 420 and 480 nm and less than half at 640 nm. The response was apparently associated with changes in carotenoid pigments. Shinohara et al.[258] reported the effect of light quality on the response of tobacco (H-mutant) exposed to 785 $\mu g/m^3$ (0.40 ppm) ozone for 30 min. The least injury occurred in far red light, followed by blue, green, and white, with the greatest effect in the red spectrum.

Photoperiod — Physiological control over some aspects of plant development is exerted by a specific light period based on a 24-hr cycle. Research has shown that plants are more sensitive to ambient oxidants and ozone when grown under an 8-hr photoperiod compared to either a 12- or 16-hr photoperiod.[119,149,176]

Light Intensity — The intensity of light affects many physiological processes within plants and is known to affect the response of plants to oxidants. Studies have been reported on the effects of light intensity before, during, and after exposure to oxidants.

Dugger et al.[73] found a direct correlation between the sensitivity of pinto bean to PAN and increasing light intensity. Pinto bean required light before, during, and after a PAN exposure for injury to occur.[72,73] This was not true of response to ozone, although some light period was necessary.

Generally, plants are more sensitive to ozone when grown under a short photoperiod at low-light intensities. This was demonstrated with pinto bean[74,119] and tobacco.[74,119,258]

Temperature — The response of plants to ozone varies with temperature. The effect of temperature on plant sensistivity to ozone varies with plant species, and thus no constant pattern of response is discernible. Some plants (radish[7,225]) were more sensitive to ozone if grown under cool conditions, and others (snap bean,[5] soybean,[75] Bel W_3 tobacco,[193,257] Virginia pine,[55,59] and white ash[309] were more sensitive if grown under warm conditions. Pinto bean[73] was sensitive to ozone regardless of temperature. Macdowell[176] found that a low day or high night temperature increased the susceptibility of White Gold tobacco to ozone. Juhren et al.[149] used eight combinations of day and night growth temperatures with *Poa annua* and then exposed these plants to ambient oxidants for a day. The sensitivity varied with plant age and was greatest at the 26° to 20°C day/night temperatures. Both high and low temperatures during growth could, in certain plants, produce physiological changes that are associated with stress resistance and thus protect the plant from air pollutant stress. Difficulties arise in interpreting the results of environmental studies conducted under greenhouse conditions.

Relative Humidity — Davis[55] and Davis and Wood[59] found an increase in sensitivity of Virginia pine to ozone at high humidity during exposure, but they reported no effects from post-exposure changes in humidity. Otto and Daines[233] found similar results for pinto bean and tobacco exposed over a wide humidity range and at several ozone concentrations. They did not study growth or post-exposure conditions. The studies of Dunning and Heck[74] showed a significant increase in the response of pinto bean, but not of tobacco, when humidity was increased during ozone exposure. Pinto bean grown at 60-percent relative humidity was more sensitive to ozone when compared to plants grown at 80-percent humidity. The response of tobacco to ozone was unaffected by humidity differences during exposure; however, its sensitivity was conditioned by the humidity before exposure. Sensitivity of tobacco decreased with an increase in humidity. Response of pinto bean was unaffected by humidity when grown under a light intensity of 4000 ft-c (43,060 lx), but it was increased with an increased humidity (80 percent) and a decrease in light intensity levels of 2000 ft-c (21,530 lx) during growth.[74] Table 11-8 summarizes some of these results.

Carbon Dioxide — Stomatal opening is decreased by high carbon dioxide levels and may affect plant sensitivity to oxidants. Heck and Dunning[119] reported a decrease in sensitivity of tobacco to ozone if the tobacco was exposed to elevated carbon dioxide concentrations of 500 ppm immediately before and during exposure to ozone (22 percent injury with added carbon dioxide, and 66 percent injury without).

METEOROLOGICAL PARAMETERS

Canadian workers using an ambient oxidant dose have correlated variations in meteorological parameters with plant injury.[179,213] A correlation was discovered when an empirical relationship involving evapo-transpiration (the coefficient of evaporation) was developed and used to modify the dose information. This empirical relationship has been used on a limited basis to predict damaging oxidant concentrations from monitored meteorologic conditions.

Linzon[172,173] also noted plant injury that was associated with changes in meteorological conditions. He reported needle blight symptoms on white pine subsequent to several days of wet weather followed by a continuous sunny period. Symptoms were observed several times during the 1957-64 growing seasons at Chalk River, Ontario. However, the time of occurrence did not correlate well with peak oxidant concentrations.

Skelly et al.[259] associated post-emergence acute tipburn on white pine growing at three different sites along the Blue Ridge Parkway with high oxidant during the summers of 1975 and 1976. The two episodes were associated with meteorological conditions that resulted in the transport of oxidant or oxidant precursors from many miles away. The episode of July 1975 resulted because of a low-pressure system (Hurricane Amy) that was off the Atlantic Coast in the vicinity of New York and a high-pressure system from Canada that became stationary over the Great Lakes. The circulation of air around a low-pressure system is counter clockwise, and it is clockwise around a high-pressure system. This pattern forced air from the northeast south into Virginia. The movement of the high-pressure system into Virginia permitted further oxidant synthesis. The June 5-12, 1976, episode resulted because of winds from the northeast on June 5 and 6 and a stationary high-pressure system that continued through June 12. As a result of the air movement, oxidant levels in July 1975 and again in June 1976 exceeded 160 $\mu g/m^3$ (0.08 ppm). In July 1975, needle injury was associated with a 1-day episode, and from June 5-13, 1976, the three sites exceeded 160 $\mu g/m^3$ (0.08 ppm) for 110, 186, and 122 consecutive hours, respectively.

A widely held belief is that vegetation growing in the humid eastern United States would be severely injured if oxidant concentrations reached the daily peak concentrations of 390 to 780 μg/m^3 (0.20 to 0.40 ppm) commonly experienced in the less humid sections of southern California. An air pollution episode that occurred on July 27-30, 1970, in the Washington, D.C., area (as well as those decribed above by Skelly et al.[259]) is indicative of what may happen. During this 4-day period, the peak oxidant concentrations ranged from 220 to 400 μg/m^3 (0.14 to 0.22 ppm) and were accompanied by a low concentration of sulfur dioxide (0.04 ppm). Oxidant injury was observed on 31 tree, 15 shrub, and 18 herbaceous species in an area of about 187 km^2 (72 miles^2). Increased emissions of the precursors associated with oxidant fomation could result in repeated occurrences of acute injury or even chronic injury to eastern vegetation.[5]

Such factors as windspeed and atmospheric pressure appear to play little or no role in affecting plant sensitivity to oxidant pollutants. Air movement would be expected to play a role under ambient conditions because of its known effect on air boundary layers of leaves, but it probably has little effect in chamber work unless wind velocities are greater than 1.6 km/hr (1 mph).[29,112,128]

EDAPHIC CONDITION

Khatamian et al.[153] reported that tomato plants grown under water stress, which did not in itself cause a reduction in growth, were more tolerant of ozone injury. The effects of soil moisture on the response of selected plants to ozone are presented in Table 11-9. Markowski and Grzesiak[186] found that bean and barley grown under drought conditions were protected from ozone injury. Several field studies[67,268,304] showed that there was a close positive correlation between soil moisture and oxidant injury to several cultivars of tobacco. Field observations generally suggest that

TABLE 11-8. RESPONSE OF PLANTS TO OZONE, AS CONDITIONED BY HUMIDITY DURING GROWTH AND EXPOSURE

Plant species	Ozone concentration, ppm, hr	Notes [a]	Growth or exposure[b]	Response, % injury (% RH)[c]				Reference
Pine, Virginia	0.25, 4	Control conditions;		(60)	(85)			59
		3-year seedlings	Exposure	4	25			
			Growth	50	58			
	0.25, 4	Juvenile	Exposure	1	35			
Bean, cultivar Pinto, and Tobacco, cultivar Bel W_3, averaged	0.40, 1	Control conditions; PP, 8 hr		(45)	(60)	(75)	(90)	233
			Growth					
			45% EH	36	39	41	31	
			90% EH	73	67	81	80	
		Control conditions; PP, 8 hr, 75% GH	Exposure	41	53	70	81	
Bean, cultivar Pinto	0.40, 1	Control conditions;		(60)	(80)			74
		PP, 8 hr; 2,000 ft-c (21,529 lx) control conditions; PP, 8 hr	Growth	66	78			
			Exposure	52	67			
Tobacco, cultivar Bel W_3	0.40, 1	Control conditions; PP, 8 hr; (2,000 ft-c (21,529 lx) control conditions; PP, 8 hr	Growth	42	36			74
			Exposure	33	36			
Tobacco, cultivar Bel W_3	0.30, 1.5	Control conditions; 31°C		(26)	(51)	(95)		233
			Exposure	9	39	50		
Bean, cultivar Pinto	0.20, 1.5	Control conditions; 31°C	Exposure	0	0	55		233
Ash, white	0.25, 4	Control conditions;		(60)	(80)			309
		1-yr seedlings	Growth	33	46			
			Exposure	38	41			
			Post-exposure	36	41			

[a] PP = photoperiod; GH = humidity during growth; EH = humidity during exposure; ft-c = foot candle

[b] Time of humidity treatment

[c] Humidity values are in parentheses.

sensitive plants may become resistant under drought conditions. Rich and Turner[249] found rapid stomatal closure in pinto bean during a 30-min exposure to ozone at 392 to 490 $\mu g/m^3$ (0.20 to 0.25 ppm) if plants were grown under a soil moisture stress; however, closure was slower under optimal water availability. They also reported rapid stomatal closure in pinto bean and Bel W_3 tobacco if the plants were conditioned and exposed to ozone at a relative humidity of 37 percent; however, a similar reponse was not found at a relative humidity of 73 percent. This evidence suggests that ozone may induce more rapid closure of stomata when plants are already under some type of water stress.

Starkey[265] found that the foliage of bean that was sensitive to subacute concentrations of PAN was also injured by drought following PAN exposure. Plants resistant to PAN were not injured similarly by drought following PAN exposure.

Salinity and low soil moisture have been shown to increase the resistance of plants to ambient oxidants.[138,139,175,222,223] Salinity, however, suppresses plant growth so that the protective effect is offset. The protective effect against ozone is enhanced with increasing salinity.[34]

The interaction of soil fertility and the response of plants to oxidant is not well understood. Nitrogen nutrition has received some attention. Menser and Street,[196] in an ambient oxidant study, reported an increase in tobacco fleck with increasing applications of soil nitrogen. This response was also found in spinach exposed to ozone.[34] Leone et al.[170] reported that concentrations of nitrogen optimal for growth resulted in tobacco being most sensitive to ozone, whereas either higher or lower nitrogen applications increased resistance. The opposite was reported for White Gold tobacco, in which the optimal nitrogen concentration for growth gave the greatest resistance to ozone.[176] Ormrod et al.[225] found that concentrations of nitrogen had no effect on growth of radish exposed to ozone. It is apparent that the nitrogen concentrations and other experimental conditions used in these studies were not sufficiently critical for an evaluation of nitrogen-oxidant interactions.

Several studies have explored the importance of phosphorus in modifying the sensitivity of plants to oxidant. Ripaldi and Brennan[252] reported an increase in phosphorus within leaves of pinto bean after exposure to ozone. Leone and Brennan[169] reported increases in ozone injury and in phosphorus content of tomato leaves with increasing applications of phosphorus. Brewer et al.[34] found an interaction between potassium and phosphorus in the response of spinach to ozone. They reported that at low phosphorus content, an increase in potassium tends to increase injury, but at high concentrations of phosphorus, the increase in potassium tends to inhibit injury. Dunning et al.[75] found that pinto bean and soybean were more

TABLE 11-9. EFFECTS OF SOIL MOISTURE ON RESPONSE OF SELECTED PLANTS TO OXIDANT STRESS[217]

			Response, percent reduction from control			
			Moisture conditions[a]			
Plant species	Oxidant concentration ppm, hr	Type of response	high	to	low	Reference
Tobacco, cultivar Catterton	Ambient oxidant		Irrigated	Normal		196
		% injury	29	11		
Tomato, cultivar Fireball			90% turgid	80% turgid		
	1.00, 1.5	% red. in chlorophyll	54	10		6
	1.00, 1.0	% red. in chlorophyll	67	24		153
	0.50, 1.0	% red. in chlorophyll	36	+3		153
	1.00, 1.0	% red. leaf dry wt	48	+40		153
Beet, garden			-40 kPa	-440 kPa	-840 kPa	223
	Control	% red. in dry wt of storage root from nonsaline control	0	24	68	
	0.20, 3 (daily for 38 days)		40	52	69	
Bean, cultivar Pinto			-40 kPa	-200 kPa	-400 kPa	139
	Control	% red. in shoot dry wt from nonsaline control	0	18	78	
	0.15, 2/day (63 days)		27	42	87	
	0.25, 2/day (63 days)		93	91	88	
	Control	% reduction in root dry wt from nonsaline control	0	25	65	
	0.15, 2/day (63 days)		25	28	78	
	0.25, 2/day (63 days)		91	89	79	

[a] Special soil moisture conditions are underlined. kPa = kilopascals (bars × 10^2)

sensitive to ozone at low potassium concentrations. Adedipe et al.[5] reported that low sulfur concentrations increased the response of Blue Lake snapbeans to ozone. McIlvenn et al.[190] reported increased injury with increasing concentrations of zinc in the soil. Soil nutrition probably plays an important role in the response of plants to pollutants only in cases of nutrient imbalances, although total nutrient salts could affect response under some conditions. In soils with balanced fertilizer regimes, plants with similar genotypes would probably respond fairly uniformly to oxidant stress. The responses of plants to ozone stress when grown under various nutrient regimes are summarized in Table 11-10.

POLLUTANT INTERACTIONS

Oxidants are components of a complex mixture of gases in the atmosphere, many of which may be phytotoxic. Except for ambient air studies and studies simulating photochemical oxidants, there have been few investigations of the effects of pollutant combinations on vegetation.

Ozone and sulfur dioxide mixtures are of special interest, owing to their widespread co-occurrence and to the possibility of a greater than additive

TABLE 11-10. EFFECTS OF VARIOUS NUTRIENTS ON RESPONSE OF SELECTED PLANTS TO OZONE (OXIDANT) STRESS[217]

Plant species	Oxidant concentration, ppm, hr	Type of response	Element	Response, % reduction from control nutritional levels[a]				Reference
				Low		to	High	
Tobacco, cultivar White Gold	0.35, 48			0.1	0.5	1.0	5.0[b]	176
		% injury	Nitrogen	21	16	4	22	
Tobacco, cultivar Catterton	Ambient oxidant			60 lb/acre		90	120	196
		% injury	Nitrogen	29		20	11	
Tomato, cultivar Rutgers	0.18, 4			28 mg/liter	280	560	1120	170
		% injury	Nitrogen	20	40	40	20	
Spinach, cultivar Viroflay	0.25, 9/day 3 days			10 ppm	30 ppm	90 ppm		34
		% injury	Nitrogen	3	18	22		
Radish, cultivar Cavalier	0.25,4			60 mg/liter	300 mg/liter			225
		% growth reduction	Nitrogen	50	55			
Tomato, cultivar Rutgers				1.5 ppm	15.5 ppm	62 ppm		170
	0.15, 3	% injury	Phosphorus	0	0	40		
	0.25, 3	% injury	Phosphorus	20	40	60		
	0.30, 3	% injury	Phosphorus	20	60	60		
Spinach, cultivar Viroflay	0.25, 9/day 3 days	% injury	Phosphorus	20 ppm	150 ppm			34
			5 ppm K	14	13			
			40 ppm K	25	9			
Radish, cultivar Cavalier	0.25, 4			30 mg/liter	150 mg/liter			225
		% growth reduction	Phosphorus	35	43			
Bean, cultivar Blue Lake	0.50, 2 day 2 days			1.3 mg/liter	32 mg/liter			5
		% chlorophyll reduction	Sulfur	55	11			
Soybean, cultivar Dare	8 doses (acute)			105 mg/liter	710 mg/liter			75
		% injury	Potassium	40	23			
Bean, cultivar Pinto	8 doses (acute)	% injury	Potassium	26	18			75

[a] Nutritional values are underlined and show a number of different units

[b] Relative to a full-strength nutrient solution

effect on vegetation.[191] Most studies of the effects of ozone on vegetation have been directed primarily to understanding the responses of various plant species or cultivars to a single air pollutant and the manner in which these responses are modified by environmental factors. However, in the field, vegetation is always exposed simultaneously to several air pollutants. When the effect produced by exposure to a combination of pollutants results in a greater effect than exposure to a single pollutant, the effects are synergistic. Antagonism is the opposite reaction. The effect level resulting from exposure to the combined pollutants is less than the sum of the injury produced by exposure to the individual pollutants. If the effect level resulting from exposure to the combined pollutants is not significantly different from the sum of the levels of the effects from individual exposures, no interaction has occurred and the foliar response to the combined pollutants is additive. Menser and Heggestad in 1966[191] first reported that exposure of the sensitive Bel W_3 tobacco to mixtures of sulfur dioxide (1310 $\mu g/m^3$ or 0.50 ppm) and ozone (59 $\mu g/m^3$ or 0.03 ppm) for 2 or 4 hr caused foliar injury of 23 to 48 percent, whereas the same concentrations of the individual gases produced no injury. The synergistic interaction between ozone and sulfur dioxide depicted in the above study stimulated plant scientists to begin research on pollutant combinations. Middleton et al.,[202] working with ratios of sulfur dioxide to ozone of 4:1 to 6:1, did not observe an increase in injury, although they found that at 4:1, ozone appeared to interfere with the expected sulfur dioxide injury. Macdowall and Cole[178] reported that the two-gas combination lowered the threshold concentrations for injury of tobacco (cultivar White Gold) by sulfur dioxide but not the threshold for ozone injury. Injury from ozone alone was first noted at a dose of 98 $\mu g/m^3$ (0.05 ppm) for 1 hr. From SO_2 alone, injury was noted at 260 $\mu g/m^3$ (0.1 ppm). And when the two gases were combined, injury was noted at the end of 1.5 hr. Macdowall et al.[179] defined the threshold in terms of dose when they reported the threshold at 20 pphm-hr (392μg-hr/m^3, or 0.20 pphm-hr). This has not appeared to be true in some other reports.[191,292] Symptoms observed when sulfur dioxide was below the threshold for SO_2 injury for the specific plant are similar to those reported for ozone.

Tingey et al.[292] exposed 11 species of plants to different ratios of sulfur dioxide and ozone mixtures. Plants were exposed to either 98 or 196 $\mu g/m^3$ (0.05 or 0.1 ppm) ozone and to 260, 660, or 1310 $\mu g/m^3$ (0.1, 0.25, or 0.5 ppm) sulfur dioxide for 4 hr. There was no general trend observed in the manner in which the ratios of pollutant concentrations caused foliar injury. Additive, greater than additive, and less than additive responses were noted (Table 11-11). Menser et al.,[194,195] Grosso et al.,[97] and Hodges et al.[134] determined the response of several *Nicotiana* species and various *N. tabaccum* cultivars to sulfur dioxide and ozone mixtures. They found that ozone and sulfur dioxide acted synergistically and produced ozone-type symptoms on all cultivars of Maryland tobacco. When plants were fumigated for 4 hr with 60 to 70 $\mu g/m^3$ (0.03 to 0.035 ppm) ozone alone or with 115 to 130 $\mu g/m^3$ (0.045 to 0.05 ppm) with SO_2 alone, no injury was observed. However, when the gases were combined and the plants were exposed for the same length of time, the result was leaf injury ranging from 5 to 15 percent. Jacobson and Colavito[146] found that sulfur dioxide at 79 $\mu g/m^3$ (0.04 ppm) decreased the sensitivity of bean to ozone and increased that of tobacco during a 4-hr exposure.

TABLE 11-11. SUMMARY EFFECTS OF SULFUR DIOXIDE AND OZONE MIXTURES ON FOLIAR INJURY[292]

	Response at concentration ratio, SO_2/O_3, ppm[a]			
Plant species	0 50/0 05	0 50/0 10	0.10/0.10	0 25/0 10
Alfalfa	–	+	+	+
Broccoli	+	0	+	0
Cabbage	0	+	0	0
Radish	0	+	+	+
Tomato	0	0	–	0
Tobacco, Bel W_3	+	+	0	+

[a] + = greater than additive; 0 = additive; – = less than additive

Differential susceptibility of individual clones of eastern white pines to ozone and sulfur dioxide was shown by Berry and Heggestad[16] and Costonis.[46] When Dochinger et al.[70] determined that chlorotic dwarf could be caused by an interaction of ozone and sulfur dioxide, they used a chlorotic-dwarf-susceptible clone to eliminate the genotype variable. Houston[140] tested the response of tolerant and susceptible clones of eastern white pine (on the basis of symptom expression under ambient conditions) to ozone or sulfur dioxide. Injury caused by sulfur dioxide or sulfur dioxide with ozone correlated well with the earlier field responses, but ozone alone did not produce a consistent response. Dochinger et al.[70] and Houston[140] found that the SO_2-O_3 mixture increased the amount of injury, but Costonis[47] reported less injury from the mixture than from sulfur dioxide alone. Both Costonis and Houston reported effects from sulfur dioxide and its mixture with ozone at concentrations of both gases well below those reported by others. It is possible that they used ultra-sensitive clonal materials. Whatever the reason, this work needs verification. Applegate and Durrant[10] also reported injury to peanut at sulfur dioxide/ozone concentrations and ozone concentrations well below those reported for other plants. Their work also requires substantiation. In the latter two cases, the concentrations reported are close to the detection limits of the gas-measuring instruments used.

Tingey et al.[287] found an additive inhibition of top growth of radish and a less-than-additive inhibition of root growth after exposure to sulfur dioxide-ozone mixtures. Tingey and Reinert[290] and Tingey et al.[293] exposed soybean, tobacco, and alfalfa to mixtures of sulfur dioxide and ozone and reported a greater-than-additive inhibition of root growth of soybean, an additive inhibition for tobacco, and a less-than-additive inhibition for alfalfa. Heagle et al.[109] reported a greater-than-additive effect on growth and yield in soybean grown under field conditions in a mixture of these gases, but the differences between the mixture and the ozone treatments were not significant. Weber[307] reported a reduction in the growth of leaves, stems, and roots of soybeans and an increase in leaf abscission as influenced by a mixture of ozone and sulfur dioxide. These changes were similar to those caused by ozone alone.

Combinations of other pollutants with ozone, PAN, or both may be important, but they have received little study. Matsushima[187] reported additive foliar effects on pinto bean and tomato from a mixture of sulfur dioxide and PAN and a less-than-additive effect on tomato from mixtures of ozone and nitrogen dioxide. Fujiwara[90] reported a greater-than-additive effect on pea from a mixture of ozone and nitrogen dioxide. Kress[161] and Kohut[157] studied the response of hybrid poplar to ozone-PAN mixtures. Kress used sequential exposures and found a greater-than-additive effect after most exposures. After others, he reported mixed responses. Kohut used simultaneous exposure and found all three types of responses in replications of the study. The reasons for these variations are unclear.

Fujiwara[90] and Reinert et al.[242] have reviewed the subject of pollutant interaction. Reinert et al.[242] tabulated some of the studies dealing with pollutant interaction. This information is presented in Tables 11-12 and 11-13.

Studies of pollutant interactions are preliminary. It is still not possible to define adequately the potential impact of pollutant combinations on the production of quality food, feed, and fiber. Plant species are known to respond differently to combinations of pollutants, and the responses can be additive, greater than additive, or less than additive. The variation in cultivar or species response or the variation in the response of plants grown or exposed under a variety of enviromental stresses is still not understood.

POLLUTANT-PARASITE INTERACTIONS

Infection by biotic pathogens has been shown to be a factor in the response of vegetation to oxidants (primarily ozone). Such responses have been studied from several perspectives since Yarwood and Middleton[314] accidentally found that rust-infected bean leaves were less sensitive to photochemical oxidants. A number of investigators have studied the protection from ozone injury afforded to plants with active infections. Others have noted that ozone injury increases the sensitivity of plants to infection. Some investigators have studied the effects of ozone on pathogens, and several have found no interacting effects. Other studies have involved bacteria, fungi, viruses, insects, and nematodes. Stark et al.[284] and Miller et al.[207] in California reported that oxidant (ozone) injury to ponderosa pine predisposed the trees to subsequent invasion by pine bark beetles. The beetles increase the rate of tree decline and may be the final cause of tree mortality[43] (Chapter 12). It is possible that oxidant stress in other parts of the country contributes to insect infestation in the forest areas (e.g., the Blue Ridge Parkway, Va.[259]). Weber[308] has shown that ozone and mixtures of ozone with sulfur dioxide (0.25 ppm, 4 hr/day) can cause a decrease in the populations of several species of nematodes parasitizing roots of soybean or leaves of begonia. These types of interactions may be of significance in areas of the country with significant oxidant pollution problems. In general, it is felt that the interacting effects resulting in modification of pathogens are due to changes in the host physiology and not to direct effects on the pathogen. Heagle[106] has reviewed pollutant-parasite interactions and has also discussed the direct effects of pollutants on pathogens themselves.

OTHER FACTORS

Heagle and Heck[111] found that Bel W_3 tobacco was predisposed to oxidant injury by previous exposure to ambient pollutants. Macdowall[176] reported the same results when the first and second exposures were to low oxidant levels. Antagonism was noted when both exposures were to high doses.

Although there has been considerable interest in understanding how various factors affect the response of plants to pesticides, especially herbicides, very little has been done with the effects of interaction between pesticides and air pollutants. Hodgson and associates[135-137] first showed an effect of ozone on the metabolism of herbicides. They found that ozone inhibited the dealkylation of atrazine in corn and altered the pathway of diphenamid metabolism in tomato. The changes could be beneficial if oxidants increase pesticide degradation, or harmful if oxidants stop the biologic breakdown at a toxic intermediate or retard the degradation process. Carney et al.[39] reported that the herbicide pebulate in combination with ozone caused a greater-than-additive effect on White Gold tobacco and that

TABLE 11-12. FOLIAR RESPONSE OF SELECTED PLANTS TO SULFUR DIOXIDE AND OZONE MIXTURES[26]

Plant species	Concentration ratio, SO_2/O_3, ppm	Exposure duration, hr	Foliar injury, %	Response to mixture[a]	Reference
Bean, garden	1.70/0.19	0.5	24	+	187
Bean, lima	0.25/0.05	4	0	0	292
Broccoli	0.50/0.05	4	17	+	292
Cabbage	1.00/0.10	4	28	0	292
Tomato	0.10/0.10	4	10	–	292
Radish	0.50/0.10	4	50	+	292
Alfalfa	0.50/0.10	4	60	+	292
Eastern white pine	0.025/0.05	6	26	+	140
Tobacco, cultivar Bel W_3	0.25/0.03	4	41	+	191
Tobacco, cultivar Bel W_3	0.50/0.10	4	88	+	292
Tobacco, Md (6 cultivars)	0.50/0.10	2	20	+	195

[a] + = greater than additive, 0 = additive, – = less than additive

TABLE 11-13. GROWTH RESPONSE OF SELECTED PLANTS TO SULFUR DIOXIDE AND OZONE MIXTURES[26]

Plant species	Concentration ratio, SO_2/O_3, ppm	Exposure duration, hr	Plant response, % reduction from control[a]	Response to mixture[b]	Reference
Radish	0.05/0.05	8/day, 5 days/week, 5 weeks	10 TDW 55 RDW	0 –	287
Radish	0.45/0.45	4	16 TDW 70 RDW	0 0	242
Alfalfa	0.05/0.05	8/day, 5 days/week, 12 weeks	18 TDW 24 RDW	– –	242
Soybean	0.05/0.05	7/day, 5 days/week, 3 weeks	24 RFW	+	293
Soybean	0.10/0.10	7/day, 5 days/week until harvest	72 TFW 63 seed wt	0 0	109
Tobacco	0.05/0.05	7/day, 5 days/week, 4 weeks	32 RDW 49 RDW	0 0	242

[a] TDW = top dry wt; RDW = root dry weight; RFW = root fresh weight; TFW = top fresh weight

[b] + = greater than additive, 0 = additive, – = less than additive ;

chloramben did the same with Delhi 34 tobacco. They reported a less-than-additive response of both tobacco cultivars to the combination of benefin and ozone. These and other herbicides acted independently of ozone exposure on tomato and white bean. Ordin et al.[224] found that *Avena coleoptile* growth was less inhibited by PAN when 2,4-D was used in amounts giving optimal growth. These interactions with herbicides need additional investigation to determine whether the responses noted are of general importance. Research needs to be directed at possible interactions between atmospheric pesticides (vapors or fine particles) and oxidant air pollutants.

Another interaction has recently been reported between cadmium applied to soil and ozone exposure of cress.[53] If cadmium potentiates the ozone response of cress, possibly other heavy metals can cause a similar interactive response.

RESPONSES OF MOSSES, FERNS, AND MICROORGANISMS

Mosses, ferns, and lichens have not been extensively studied to determine ozone and oxidant effects. Comeau and LeBlanc[45] found that a 4-hr exposure of *Funaria hygrometrica* to ozone at 490 to 1960 μg/m^3 (0.25 to 1.00 ppm) stimulated the regenerative capacity of the moss leaves. This did not occur with 6- and 8-hr exposures. Glater[95] reported oxidant injury to several species of fern growing in the Los Angeles area. Initially, tan colored lesions appeared near the smaller veins, but in no special pattern. Later, the entire leaf became necrotic. Symptom development and sensitivity of leaves were different from those noted in more highly evolved plants. All leaves appeared to be equally sensitive, except for the growing tip and the youngest uncoiling leaves. Occasionally, a young plant was killed.

Lichenologists have long used the presence and abundance of lichen species to map the biologic impact of large urban and industrial areas.[88] Though early workers considered changes in the presence and abundance of these organisms to be related to such factors as temperature and humidity, more recently, most researchers have tended to relate changes in lichen population more to industrial air pollution than to other environmental changes. There is strong indication that both the presence and the abundance of certain lichen species are correlated with sulfur dioxide concentrations in urban and industrial areas. However, little is known about the direct effects of ozone or other oxidants on lichen morphology or physiology. Ozone or some of the other oxidants may have an adverse ecological impact on lichens.

Ozone in high concentrations has been used in a variety of applications for the control and suppression of fungi and bacteria. These applications have included food protection, drinking-water purification,[99] and treatment of sewage.[154,188] The germicidal effectiveness depends on concentration, relative humidity, and the specific organism. In many cases, even a concentration of 5880 to 9800 μg/m^3 (3 to 5 ppm) was not sufficient to kill some bacteria. Burleson et al.[36] showed inactivation of several viruses and bacteria after ozone exposure, and a greater inactivation with simultaneous sonication. Zobnina and Morkovina[315] related the tolerance of a carotinoid strain of *Mycobacterium carothenum* with the presence of the pigment.

Large doses of ozone may inhibit growth and sporulation of fungi on fruit, although most fungi tested were resistant to ozone. Spalding[262] reported that ozone acted as a surface biocide. Above 980 μg/m^3 (0.5 ppm), ozone inhibited surface growth of fungi on strawberry and peach. Ridley and Sims[251] extended the shelf-life of strawberry and peach by exposing them to ozone, but stated no concentrations. Ozone at 1960 to 3720 μg/m^3 (1 to 2 ppm) for 1 to 2 hr/day controlled the surface growth of fungi and sporulation on apple, reduced offensive odors, and decreased the ripening rate.[261] Watson[305] found that ozone at 785 to 3920 μg/m^3 (0.4 to 2.0 ppm) acted as surface fungicide on fruit. Fungal growth within the fruit was not affected. Sporulation and some control of decay by *Penicillium digitatum* and *P. italicum* were noted in open storage boxes of lemon and orange exposed to ozone at 1960 μg/m^3 (1 ppm).[51] Ozone was more effective than a fungicide dip in controlling *Botrytis* bud rot of gladiolus, but no concentrations were given.[181] In general, researchers have suggested that rather large dosages of ozone are required to protect storage fruits from fungal infection. These concentrations may be so high as to preclude the use of ozone in storage facilities. The ability of ozone to reduce spore germination in fungi apparently depends on species, spore morphology, moisture, and substrate.[106] Single-celled spores and those with thin cell walls are most sensitive. Wet spores are more sensitive than dry spores.

Hibben[127] found ozone toxic to moist fungus spores of some species, even at concentrations of 200 μg/m³ (0.1 ppm). Exposure to 980 and 1960 μg/m³ (0.5 and 1.0 ppm) reduced or prevented germination of spores of all species tested. Ozone at 200 μg/m³ (0.1 ppm) for 4 hr or at 1960 μg/m³ (1.0 ppm, for 2 hr stopped apical cell division of conidiophores of *Alternaria solani* and caused collapse of the apical cell wall.[248]

Heagle[106] has reviewed the effects of ozone on fungus growth, sporulation, and germination. Ozone may inhibit colony growth on artificial media, but it rarely causes death, even at high concentrations. Differences in species susceptibility are known. In several fungi, exposure to ozone at 196 or 785 μg/m³ (0.10 or 0.40 ppm) for 4 hr caused a 10- to 25-fold increase in sporulation.[106] Heagle[107] also reported the effects on three obligate parasitic fungi of low exposures to ozone. Germination of spores was not affected in any of these studies. Kuss[162] grew 30 representative fungi on agar and found that spore production in some species was increased after exposure to ozone.

Rabotnova et al.[240] exposed two species of yeast to ozone: *Candida lipolytica* was sensitive, and *C. auilliermondii* was resistant. The biocidal activity of ozone was determined under various cultural conditions with air streams of about 294,000 to 10,780,000 μg/m³ (150 or 5500 ppm) (v/v for 10 to 30 min). Ozone was an effective biocide under most conditions. Its effectiveness increased with decreasing pH of the culture medium. Kanoh[151] found that exposure to ozone at 58,800 μg/m³ (30 ppm) for 30 min increased oxygen uptake in slime mold homogenate from *Physarum polycephalum*. Ozone also increased succinoxidase activity and inhibited part of glycolysis. Effects of ozone on *Euglena gracilis* that were reported by de Koning and Jegier[61-64] included reduction of net photosynthesis, increase in respiration, and effects on pyridine nucleotide reduction and phosphorylation. They also reported that the reduction of net photosynthesis was a logarithmic function of ozone concentration in 1-hr exposures. These investigators also found a 5-percent reduction in oxygen evolution after a 1-hr exposure to 980 μg/m³ (0.5 ppm) ozone bubbled into 5 ml of solution and an additive effect with a mixture of sulfur dioxide and ozone.[63] Verkroost[303] carried out a detailed study of the effects of ozone on *Scenedesmus obtusiusculus*, Chod. with special concern over the effects on photosynthesis and respiration. A major weakness in this study was the use of an air stream containing ozone at 58,800 μg/m³ (150 ppm). The report suggested that the primary site of ozone action is the membrane structure, which produces changes in photosynthesis and respiration.

Giese and Christiensen[94] found that protozoa in hanging drop suspensions exposed to approximately 8 percent ozone in ozonized water were killed within a period of 4 min (*Colpidium*) to a maximum of 64 min (*Tillina*).

Haines[102] reported that ozone at 4 ppm retarded the growth of *Escherichia coli*, whereas 19,600 μg/m³ (10 ppm) prevented growth. Scott and Lesher[254] found that approximately 2 x 10^7 molecules of ozone per bacterium killed 50 percent of the cells of *E. coli* and that the primary effect was on the cell membrane. Elford and van den Ende[76] reported that ozone at 390 μg/m³ (0.2 ppm) had a lethal effect on some bacteria deposited from aerosol mists on various surfaces. Relative humidity is an important factor, particularly when ozone concentration is low. They found little death at a humidity below 45 percent at ozone concentrations of 1960 μg/m³ (1 ppm), as opposed to a 90-percent kill in 30 minutes at 40 μg/m³ (0.025 ppm) ozone with a humidity of around 70 percent. A 5-min exposure of *Bacillus cereus* to ozone at 0.12 mg/liter was the minimal lethal dose, whereas 0.10 mg/liter was effective for *B. megaterium* and *E. coli*.[62] Spores of the *Bacillus* sp. were killed by ozone at 2.29 mg/liter. In most of the research studying the effects of ozone on lower organisms, ozone has been used as a germicide or as part of an attempt to understand the interactions of pollutants and pathogens on higher plant response.

SUMMARY

Oxidant injury to vegetation was first identified in 1944 in the Los Angeles Basin. Our understanding of oxidant effects and of the widespread nature of their occurrence has increased steadily since then. Although the major phytotoxic components of the photochemical oxidant complex are ozone and peroxyacetylnitrate (PAN), some data suggest that other phytotoxicants are also present. The peroxyacylnitrates are the most phytotoxic of the known photochemical oxidants; however, the ubiquitous nature of ozone and its association with widespread injury to vegetation make it the most important phytotoxic component of the photochemical oxidant complex.

The effects of photochemical oxidants on vascular plants can be envisioned as occurring at several levels, ranging from the subcellular to the organismal, depending on the concentration and duration of exposure to the pollutant, and on the elapsed time after exposure that effects are observed.

The earliest effect is an increase in cell membrane permeability. Cellular and biochemical changes are ultimately expressed on the organismic level in visible foliar injury, increased leaf drop, reduced plant vigor, reduced plant growth, and death. In the final analysis, biochemical modifications on an individual plant level are manifested by changes in plant communities and then in whole ecosystems.

Leaf stomata are the principal plant entry sites for ozone and PAN. Oxidants affect such physiologic processes as photosynthesis, respiration, transpiration, stomatal opening, metabolic pools, biochemical pathways, and enzyme systems.

Visible injury is identifiable as pigmented, chlorotic, or necrotic foliar patterns. Metabolic cellular disturbances can occur without visible injury and may be reversible. However, most of the growth effects reported until recently were associated with visible injury.

Classic ozone injury is demonstrated by the upper-surface leaf fleck on tobacco and the leaf stipple of grape. Many plants show an upper-surface response with no associated injury to the lower surface of leaves. However, in monocotyledonous plants such as grasses or cereals, and some nonmonocotyledonous plants, the mesophyll tissue is not divided, and bifacial necrotic spotting (flecking) is a common symptom of ozone injury.

Coniferous trees exhibit different symptoms. Ozone is probably the cause of emergence tipburn in eastern white pine (white pine needle dieback) and chlorotic decline, a needle injury of ponderosa pine.

Classic PAN injury appears as a glaze followed by bronzing of the lower leaf surface of many plants. Complete collapse of leaf tissue can occur if concentrations are sufficiently high. Early leaf senescence and abscission usually follow the chronic response symptoms. Chronic injury patterns generally are not characteristic and may be confused with symptoms caused by biotic diseases, insect infestation, nutritional disorders, or other environmental stresses.

A great deal of research has been done to define the effects of oxidants on plant growth and yield more accurately. Studies comparing the growth of plants in filtered and nonfiltered field chambers using oxidants in the ambient air have reported up to 50-percent decreases in citrus yield (orange and lemon); 10 to 15 percent supression in grape yield in the first year, and 50 to 60 percent reduction over the following 2 years; and a 5 to 29 percent decrease in yield of cotton lint and seed in California. Losses of 50 percent in some sensitive potato, tobacco, and soybean cultivars have been reported in the eastern United States. It is apparent that oxidants in the ambient air reduce yields of many sensitive plant cultivars.

Experimental chambers with controlled environments have been used to study both short-term and long-term effects of exposure to ozone (Tables 11-2 and 11-4). Multiple acute exposures (785 $\mu g/m^3$, or 0.40 ppm) of radishes for 1.5 hr resulted in reductions in root growth. The reductions in root growth from the multiple ozone exposures were equal to the additive effects of three single exposures. When soybean plants were exposed to 1468 $\mu g/m^3$ (0.75 ppm) ozone for 1 hr, root growth was consistently reduced more than top growth. There was also a reduction in nodule weight and number. The greater reduction of root growth than top growth is related to the transport of photosynthate. Ozone also affects the process of nitrogen fixation in clover, soybean, and pinto bean through reduction in nodule number, but neither nodule size nor efficiency of nitrogen fixation is influenced. The effect of ozone on the nitrogen fixation process in legumes, if widespread, could have a major impact on plant communities and affect fertilizer requirements. There are indications that the effect of ozone on nodulation may be related to carbohydrate supply in host plants.

Experimental long-term exposures of a variety of crops as well as ornamental and native plants to ozone have resulted in a reduction in growth and/or yield. Throughout a growing season, 14 species representative of the aspen plant community were exposed to ambient air (98 to 137 $\mu g/m^3$ ozone, or 0.05 and 0.07 ppm) and to ozone (290 and 588$\mu g/m^3$ or 0.15 and 0.30 ppm) for 3 hr/day, 5 days/week, and to charcoal-filtered air. Foliar injury to all species resulted at the highest pollution concentration. The growth of two soybean cultivars

(Hood and Dare) was inhibited by intermittent exposure to ozone at 196μg/m³ (0.10 ppm) for 3 weeks. Growth of both root and top was decreased. Similar results were noted with radish, except that a lower concentration (98 μg/m³ or 0.05 ppm) inhibited growth. In the aforementioned studies, the reduced growth occurred even though there were very few visible symptoms of plant injury.

A reduction of 30 percent in the yield of wheat occurred when, at anthesis, wheat was exposed to ozone at 392 μg/m³ (0.2 ppm), 4 hr/day for 7 days. A significant reduction in the yield of tomato was noted when plants were experimentally treated with ozone at 686 μg/m³ (0.35 ppm). Lower fruit set and fewer harvested fruit caused the reduction. Chronic exposures to ozone at 98 to 290 μg/m³ (0.05 to 0.15 ppm) for 4 to 6 hr/day produced reductions in yield in soybean and corn grown under field conditions. The threshold for measurable effects for ozone appears to be between 98 and 196 μg/m³ (0.05 to 0.10 ppm) for sensitive plant cultivars. This is well within the range of ozone levels monitored in the eastern United States. Growth or flowering effects were reported for carnation, geranium, radish, and pinto bean grown in greenhouse chambers exposed to ozone at 98 to 294 μg/m³ (0.05 to 0.15 ppm) for 2 to 24 hr/day.

The two most critical factors in determining plant response to air pollution are duration of exposure and concentration of pollutants. These two factors describe exposure dose. In determining the response of vegetation to oxidants, concentration is more important than time. Any given dose presented to a plant in a short period of time has a greater effect than the same dose applied over a longer period.

The concept of limiting values was used by Jacobson to define the boundary between doses of a pollutant that are likely to cause measurable foliar injury to vegetation and those that are not. Foliar injury was used as the index of plant response. The ranges for limiting values for effects of ozone are:

1. Trees and shrubs:
   400 to 1000 μg/m³ (0.2 to 0.51 ppm) for 1 hr
   200 to 500 μg/m³ (0.1 to 0.251 ppm) for 2 hr
   120 to 340 μg/m³ (0.06 to 0.17 ppm) for 4 hr
2. Agricultural crops:
   400 to 800 μg/m³ (0.2 to 0.41 ppm) for 0.5 hr
   200 to 500 μg/m³ (0.1 to 0.251 ppm) for 1 hr
   75 to 180 μg/m³ (0.04 to 0.09 ppm) for 4 hr

The range of limiting values for PAN is:
1000 μg/m³ (0.2 ppm) for 0.5 hr
500 μg/m³ (0.1 ppm) for 1 hr
175 μg/m³ (0.035 ppm) for 4 hr

Doses of ozone or PAN greater than the upper limiting values are likely to cause foliar injury.

The data points used to determine the limiting values listed above are not necessarily threshold values but were based on available published research data. More than 200 studies were surveyed. Any limitations that were present in the experimental techniques used in the studies are expressed in the data points. Another constraint of the PAN data is the limited number of studies.

Plant sensitivity to ozone and PAN is conditioned by many factors. Genetic diversity in sensitivity to ozone between species and cultivars within a species is well documented. Variants in sensitivity to ozone within a natural species are well known for several pine species, including white, loblolly, and ponderosa. Plant sensitivity to oxidants can be changed by both climatic and edaphic factors. A change in environmental conditions can initiate a change in sensitivity at once, but it will be 3 to 5 days before the response of the plant is totally modified. Plants generally are more sensitive to ozone when grown under short photoperiods, medium light conditions, medium temperature, high humidity, and high soil moisture. Injury from PAN may increase with increasing light intensity. Conditions during exposure and growth affect the response of plants to oxidants in similar ways. In general, environmental conditions optimum for plant growth tend to increase the sensitivity to ozone. At the time of exposure, factors that increase water stress tend to make plants more tolerant to ozone. Soil moisture is probably the most important environmental factor that affects plant response to oxidants during the normal growing season. Physiologic age affects the response of the leaf to oxidants. Young leaf tissue is most sensitive to PAN, whereas newly expanding and maturing tissue is most sensitive to ozone. Light is required for plant tissue to respond to PAN; a similar light requirement is not needed for plants to respond to ozone.

The majority of effects observed, such as suppression of root growth, mineral uptake, and nitrogen fixation, apparently result from a suppression in photosynthesis and modifications in photosynthate distribution. This suppression in metabolic reserves ultimately slows plant growth and renders the plant more sensitive to other stresses. Physiological changes can provide a sensitive means of monitoring the health and vigor of the plant with or without visual injury. Ozone affects pollen germination in some species and may affect yield through incomplete pollination of flowers. Investigations with *Arabidopsis thaliana* showed no mutagenic effects from ozone over seven generations.

Mixtures of pollutants can cause effects below the levels caused by either gas alone; however, there is some disagreement concerning ozone interactions with other gases. Ratios of gas mixtures, intermittent exposures, sequential exposures to pollutants, and predisposition by one pollutant to the effects of a second pollutant may be important in nature, but insufficient knowledge is available to elucidate the effects.

The response of plants to oxidants may be conditioned by the presence or absence of biotic pathogens. Depending on the plant and the pathogen, oxidants may cause more or less injury to a given species. Oxidant injury to ponderosa pine predisposes the trees to later invasion by bark beetles. Ozone and ozone-sulfur dioxide mixtures can decrease the population of some plant-parasitic nematodes. Variable plant responses have been noted when herbicides were used in the presence of high oxidant concentrations.

Little research on the effects of oxidants on ferns, nonvascular green plants, and microorganisms has been reported. Lichens and mosses are responsive to acid gases, but there is no definite evidence that they respond to oxidants. Ferns may be especially sensitive, but their injury response is different from that of higher vascular plants. Growth and sporulation of fungi on surfaces are usually, but not always, affected. Ozone from 0.1 to several milligrams per liter of solution is required to kill many microorganisms in liquid media. Most work with microorganisms has been done to study the effectiveness of ozone as a biocide in the storage of vegetation or treatment of water or sewage supplies.

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12. ECOSYSTEMS

INTRODUCTION

The preceding chapters have dealt with the formation and chemistry of ozone and other oxidants and with the effects of oxidant/ozone on human health and on experimental plants and animals. In this chapter, the effects of oxidant-pollutant stress on simple and complex communities are discussed to illustrate that plants, animals, man, and the environment are interconnected and that the relationships between them are profoundly complex.

Plants, animals, and microorganisms usually do not live alone, but exist as populations. Populations live together and interact as communities. Commmunities, because of the interactions of their populations and of the individuals that constitute them, respond to pollutant stress differently from individuals. Man is an integral part of these communities, and as such, he is directly involved in the complex ecological interactions that occur within the communities and the ecosystem of which he is a part.

The stresses placed on the communities and the ecosystems in which they exist can be far-reaching, since the changes that occur may be irreversible. For example, it has been suggested that the arid lands of India are the result of defoliation and elimination of vegetation that induced local climatic changes not conducive to the reestablishment of the original vegetation.[10]

An ecosystem (e.g., the planet Earth, a forest, a pond, an old field, or a fallen log) is a major ecological unit made up of living (biotic) and physical (abiotic) components through which the cycling of energy and nutrients occurs (Table 12-1). A structural relationship exists among the various components. The biotic units are linked together by functional interdependence, and the abiotic units make up all of the physical factors and chemical substances that interact with the biotic units. The processes occurring within the biotic and abiotic units and the interactions among them are subject to environmental influences.[9]

TABLE 12-1. COMPOSITION OF ECOSYSTEMS

Component	Description
Biotic (biological):	
Individuals	Plants, animals (man), and microorganisms. These are either producers, consumers, or decomposers.
Producers	Green plants.
Consumers	Herbivores, carnivores.
Decomposers	Macroorganisms (mites, earthworms, millipedes, and slugs) and microorganisms (bacteria and fungi).
Populations	Groups of similar and related organisms.
Communities	Interacting populations linked together by their responses to a common environment.
Abiotic (physical):	
Energy	Radiation, light, temperature, and heat flow.
Water	Liquid, ice, etc.
Atmosphere	Gases and wind.
Fire	Combustion.
Topography	Surface features.
Geological substratum	Soil, a complex system. Nutrients.

Ecosystems tend to change with time, and sequential changes in the types of populations within a community are usually recognizable. Adaptation, adjustment, and evolution are constantly taking place as the biotic component, the populations, and the communities of living organisms interact with the abiotic component in the environment. The interaction and exchange of energy and nutrients over time result in sequential or, in some cases, cyclic or telescoped changes in populations and communities. The sequential replacement of one population by another in a continual series, going from pioneer (first and less diversified) populations to so-called climax (mature and more diversified) communities, is termed "succession."[9] Climax communities are structurally complex and more or less stable, and they are held in a steady state through the operation of a particular combination of biotic and abiotic factors. Man is often a factor, as for example, when his grazing cattle maintain a pasture.[9] The disturbance or destruction of a climax community or ecosystem results in its being returned to a simpler stage.[71,72] Existing studies indicate that changes occurring within ecosystems, in response to pollution or other disturbances, follow definite patterns that are similar even in different ecosystems. It is therefore possible to predict broadly the basic biotic responses to the disturbance of an ecosystem.[22,39,70-72] These responses to disturbances are:

1. Reduction in standing crop.
2. Inhibition of growth or reduction in productivity.
3. Differential kill (removal of sensitive organisms at the species and subspecies level).
4. Food chain disruption.
5. Successional setback.
6. Changes in nutrient cycling rates.

Organisms vary in their ability to withstand environmental changes. The ability of a population to withstand injury from polluted air, weather extremes, herbicides, or other disturbances depends on its range of tolerance—that is, the range of variation within which it can survive and function. The range of tolerance (also termed the "law of tolerance," Figure 12-1), differs because of inter- and intraspecific variations in susceptibility to injury by polluted air or other disturbances.[50] The highly specialized populations (those with a narrow range of tolerance or adaptability) are either reduced in number or eliminated.

Diversity and structure are most changed by pollution as a result of the elimination of sensitive species of flora and fauna and the selective removal of the larger overstory plants in favor of plants of small stature.[71,72] The result is a shift from the complex forest community to the less complex, hardy shrub and herb communities. The opening of the forest canopy changes the environmental stresses on the forest floor, causing differential survival and, consequently, changed gene frequencies in subcanopy species.

Associated with the reduction in diversity and structure is a shortening of food chains, a reduction in the total nutrient inventory, and a return to a simpler and less stable successional stage.[71,72] In addition, the pollutants act as predisposing agents, and an increase in the activity of insect pests and certain diseases occurs.[48,71] It

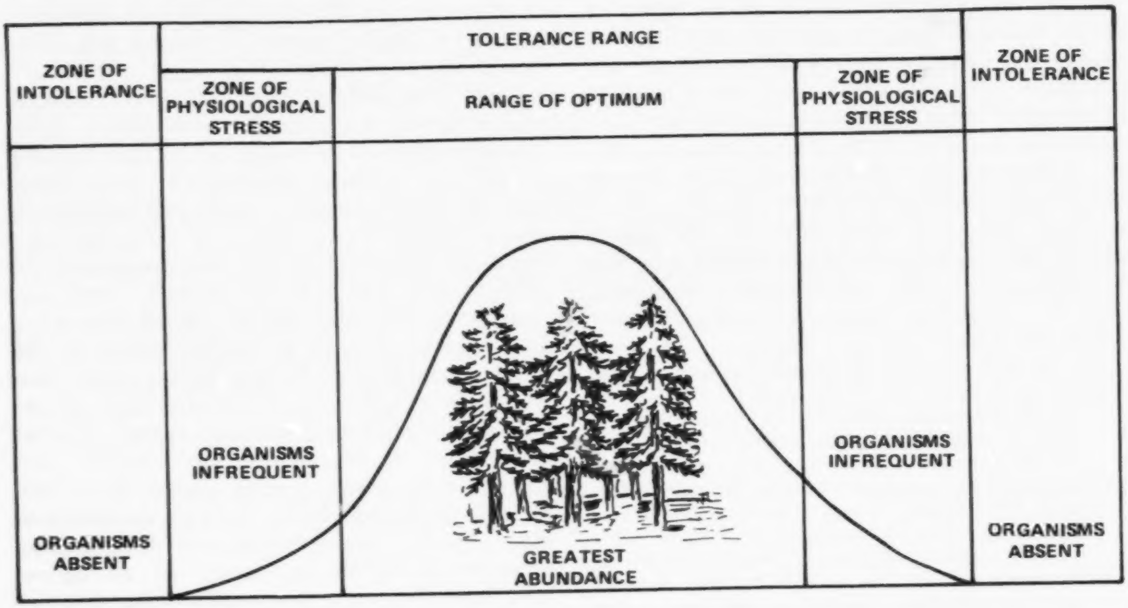


Figure 12-1. Law of tolerance.[50]

should be emphasized that ecosystems are usually subjected to a number of stresses at the same time, not just to a single perturbation.

Ecosystems are usually evaluated by modern man solely on the basis of their economic value to him. This economic value, in turn, depends on the extent to which man can manipulate the ecosystem for his own purpose. This single-purpose point of view makes it difficult to explain the many benefits of a natural ecosystem to man's welfare in terms of the conventional cost-benefit analysis. Gosselink et al.[24] have, however, placed a value on a tidal marsh by assigning monetary values to the multiple contributions to man's welfare such as fish nurseries, food suppliers, and waste treatment functions of the marsh. They estimate the total social values to range from $50,000 to $80,000/acre.

Westman[67] also evaluated the benefits of natural ecosystems by estimating the monetary costs associated with the loss of the free services (absorption of air pollution, regulation of global climate and radiation balance, and soil binding) provided by the ecosystems. Westman estimated that the oxidant damage to the San Bernardino National Forest could result in a cost of $27 million per year (1973 dollars) for sediment removal alone because of erosion as long as the forest remained in the early stages of succession.

Estimates of the dollar values of such items as clean air and water, untamed wildlife, and wilderness (once regarded as priceless) are attempts to rationalize the activities of civilization.[67] When environmental decisions are made based on the cost of damaged ecosystems, it is usually assumed that the path chosen will be that which is most socially beneficial, as indicated by costs compared to benefits. As Westman[67] points out, certain corollaries accompany the assumption that decisions maximizing the benefit: cost ratios simultaneously optimize social equity and utility. These corollaries are:

> (1) The human species has the exclusive right to use and manipulate nature for its own purposes. (2) Monetary units are socially acceptable as means to equate the value of natural resources destroyed and those developed. (3) The value of services lost during the interval before the replacement or substitution of the usurped resource has occurred is included in the cost of the damaged resource. (4) The amount of compensation in monetary units accurately reflects the full value of the loss to each loser in the transaction. (5) The value of the item to future generations has been judged and included in an accurate way in the total value. (6) The benefits of development accrue to the same sectors of society, and in the same proportions, as the sectors on whom the costs are levied, or acceptable compensation has been transferred. Each of these assumptions, and others not listed, can and have been challenged.[67]

In the case of corollary 4, for example, the losses incurred include species other than man, but these losses are seldom, if ever, compensated. Also, the public at large is not usually consulted to determine whether the dollar compensation is adequate and acceptable. Frequently, there is no direct compensation. Corollary 5 can never be fulfilled because it is impossible to determine accurately the value to future generations.

Evaluating the contribution of functioning natural ecosystems to human welfare is a very complex task and usually involves weighing both economic and human social values. However, because natural ecosystems are life support systems, their values should not be quantified in economic terms.

With the passage of time, man has destroyed many of the naturally occurring ecosystems of which he was a part and has replaced them with simplified ecosystems wholly dependent on his care and protection and requiring a large input of energy.[50]

Man favors the simple, unstable and synthetic ecosystems, because when they are extensively managed and subsidized by the use of fossil fuels, they are highly productive. Productivity is the rate at which energy is stored by the photosynthetic activity of green plants.[9,50] Young and successional ecosystems are more productive because they add biomass (accumulate energy) each year. An agricultural ecosystem (agroecosystem) is an example of such a simplified ecosystem. Urban ecosystems, with their cultivated trees, shrubs, grasses, and flowers, are also examples of ecosystems simplified by man. The structural simplicity of these ecosystems (in many cases monocultures) makes them less resistant to environmental and disease stresses or to perturbations such as oxidant pollution. The greater diversity of a mature ecosystem retards the disruption of normal structure and function caused by any type of perturbation. For example, a forest ecosystem in which the communities are composed of many species would show less immediate damage by stress than successional stages having only a few species. Even greater damage would be anticipated in an agroecosystem, which may be considered the simplest of successional stages since often only a single producer species is present.

GENERAL RESPONSES OF NATURAL AND AGROECOSYSTEMS TO STRESS BY OXIDANTS

Agroecosystems

Photochemical oxidant air pollutants have been recognized as a type of chronic pollution problem only during the last 20 to 25 years. The problem was first recognized in southern California, where it perhaps has had its greatest impact. Citrus groves and vineyards in the inland valleys of southern California are prime examples of stress to agroecosystems caused by chronic exposure to oxidants.

Studies were initiated in 1960 on lemon and navel orange trees[58] and in 1968 on wine grapes to determine the economic losses caused by oxidant pollutants. Both studies were conducted under field conditions for several years. The lemon and orange tree studies provided only a small amount of data that may be interpreted in an agroecosystem context because they were highly oriented toward the primary producer components,[58-60] and no effort was made to examine the effects of oxidants on consumer components and decomposer organisms. The studies indicated that oxidant stress reduced water use and photosynthesis, increased leaf and fruit drop, and resulted in a severe reduction in the yield of marketable fruit. All of these effects occurred without the development of plainly visible leaf symptoms. None of the above studies managed to distinguish the different effects of ozone and peroxyacetylnitrate (PAN) or its homologues in the photochemical oxidant mixtures; however, inferences drawn from these data suggest probable impact on the various components of these agroecosystems, particularly on the levels of consumer and decomposer populations. Accelerated leaf drop may influence the development of insect pests such as aphids, scale insects, and red citrus mites. Higher concentrations of amino acids or free sugars in injured leaves before abscission could result in an increase in pest populations or a diminution if leaves fall too rapidly. Leaf and fruit drop would provide an increased substrate for populations of decomposer organisms at the soil surface.

Natural Ecosystems

Natural ecosystems in California and in the Appalachian Mountains in the eastern United States have also suffered from oxidant pollution.

THE APPALACHIAN MOUNTAINS

In the eastern United States, a disease called emergence tipburn, found in eastern white pine, was related to ozone by Berry and Ripperton.[5] The disease is characterized by bands of necrosis initiated in the semimature tissue of elongating needles and spreading to the needle tip.

Under forest conditions, the affected trees occur randomly in the stand, and the same trees are injured repeatedly in a single season or in successive years.[4] Only sensitive genotypes are affected, and these are gradually eliminated. Eastern white pine either forms pure stands or occurs in mixtures with other species in abandoned fields. Under these conditions, it is an important pioneer tree.[65] In established stands, it is a major component of 4 forest types and an associate in 14 other types and inhabits a range extending over 7 million acres, from the Lake States to the Appalachian Mountains.[65] Berry[4] in 1961 reported that post-emergence tipburn occurs throughout the natural range of this species. There is also evidence of a slow decline in tree vigor caused by the deterioration of feeder rootlets. Skelly et al.[49] in 1975 and 1976 also noted post-emergence acute tipburn on white pine growing at three different sites along the Blue Ridge Parkway.

High concentrations of ozone in the forested areas of the eastern United States cause severe injury to eastern white pine[3,49,65] and other forest species. Berry[3] reported levels of oxidant as high as 230 $\mu g/m^3$ (0.12 ppm) at a mountaintop monitoring station in June 1962. An air monitoring network operated by Virginia Polytechnic Institute and State University at three locations (the Blue Ridge Mountains, the Shenandoah Valley, and the southern Appalachians) recorded total oxidant peaks as high as 250 $\mu g/m^3$ (0.13 ppm) during early July 1975, along with 43 hr of exposure at concentrations of 160 $\mu g/m^3$ (0.08 ppm) or higher for 9 days.[22] In June 1976, oxidant levels exceeded 160 $\mu g/m^3$ (0.08 ppm). After these episodes, significant increases in oxidant injury were observed (particularly in the Blue Ridge Mountains) involving three categories of eastern white pine: Those previously without symptoms, those with chlorotic mottle, and those exhibiting chlorotic dwarf symptoms. Such incidences suggest the need for more comprehensive studies of oxidant (and sulfur dioxide) effects in forests of the eastern United States.

The oxidant peaks mentioned above were related to oxidant transport from urban areas to the northeast (see Chapter 11, section on meteorological parameters).

Studies by Davis and Wood [15] suggest that other conifer species, in particular, Virginia pine and jack pine, may be more sensitive to ozone than eastern white pine. In addition, there is a synergistic interaction between low concentrations of ozone and sulfur dioxide that is the cause of the chlorotic dwarf disease of eastern white pine.[16] A study by Ellertsen et al.[19] showed 10 percent mortality between 1956 and 1965 in dominant and codominant eastern white pines near an industrialized area that included several hundred square miles on the Cumberland Plateau. The combined effects of ozone and sulfur dioxide were considered responsible for the tree decline. Because eastern white pine represented only 5 percent of the total wood volume available for harvest, the economic impact was slight. There was no effort to interpret pollutant effects in an ecosystem context. In the long run, the broader question to be addressed regards the effects of pollutant stress on all major ecosystem components, primarily on producers, consumers, and decomposers. An analysis of the multiple effects of oxidants on eastern forest ecosystems will present a much more adequate picture of the effects pollution is having on these forests.

The studies to delineate the effects of oxidant episodes in the Appalachian Mountains are continuing.

CALIFORNIA

In southern California, the coastal chaparral ecosystem, dominated by chamise and manzanita or by woodland species (including the live oaks and big cone spruce), and the coniferous forest ecosystems have received severe exposure. The desert ecosystems in the vicinity of mountain passes connecting the coastal and desert regions have also undoubtedly been exposed.

Injury has been well documented only in the mixed-conifer forest ecosystem of the San Bernardino Mountains. Early symptoms of injury in conifer species were reported in a number of California national forests in 1970.[36] In the southern Sierra Nevada, Forest Service surveys made in 1974[69] have detected increased injury in ponderosa pine at many locations in the Sequoia National Forest, Sequoia National Park, and Kings Canyon National Park since 1970. Specific stands of mixed conifer forest on the western slopes of the southern Sierra Nevada now appear to be affected by oxidants from the San Joaquin Valley.[35] The potential loss of timber growth in this area alone is a very serious prospect.

A project is now underway[55,56] to determine in detail the effects of oxidant air pollutants on a mixed conifer forest ecosystem in southern California. The planning documents and early results from this study as described in the NAS document, *Ozone and Other Photochemical Oxidants*,[38] constitute the major source of information found in the remainder of this chapter.

ORIGIN OF INJURIOUS CONCENTRATIONS OF OZONE AND OXIDANTS

Advection from Urban Centers to Remote Areas in Southern California

Descriptions of the vertical and horizontal distributions of photochemical smog in the Los Angeles Basin (southern coastal air basin) during typical summer days have been provided by Blumenthal et al.,[6] Edinger,[17] Edinger et al.,[18] and Miller et al.[34] Important observations to be drawn from their reports are the interactions of basin and mountain topography and local meteorology in determining the transport and concentrations of oxidant air pollutants in relation to elevational zones of vegetation.

The marine temperature inversion layer that frequently forms above the heavily urbanized Los Angeles metropolitan area often extends inland as far as 144 km (90 miles), depending on season and time of day. Surface heating of air under the inversion increases with distance eastward in the basin and often disrupts the inversion by midmorning at its eastern edge. The northern rim of the basin is formed by the San Gabriel and San Bernardino Mountains, interrupted only by the Cajon Pass about 88 km (55 miles) inland (see Figure 12-2). The marine temperature inversion layer encounters the mountain slopes, usually below 1200 m (4000 ft). In the morning, the temperature inversion often remains intact at this juncture, and air pollutants are confined beneath it. Studies by Edinger[17] and Edinger et al.[18] have described how the heated mountain slopes act to vent oxidant air pollutants over the crest of the mountains and cause the injection of pollutants into the stable inversion layer horizontally away from the slope. Oxidant concentrations within the inversion are not uniform, but occur in multiple layers and strong vertical gradients. In some cases, the inversion may serve as a reservoir for oxidants, principally ozone, which may arrive at downwind locations along the mountain slopes relatively undiluted because of a lack of vertical mixing within the inversion layer and a lack of contact with ozone-destroying material generated at the ground. The important result of the trapping of oxidant in these layers is its prolonged contact with high terrain at night.

The most important effect of the interaction of pollutant and inversion layer at the heated mountain slope is the vertical venting of oxidants over the mountain crest by upslope flow. A gradient of oxidant concentrations or doses is established across distinct vegetation zones ordered along an elevational gradient, i.e., the chaparral and conifer forest ecosystems that occupy the slopes and mountain terrain beyond the crest, respectively. The altitudinal sequence of these ecosystems is illustrated in Figure 12-3. According to Horton,[26] the chaparral zone is subdivided, from lower to higher elevations, into three subzones called the chamise, manzanita, and woodland chaparral; the mixed conifer forest occupies the mountain crest.

The daytime changes in oxidant concentrations at several stations (A, B, C, and D in Figure 12-2) along the southern slope and at the crest of the San Bernardino Mountains is illustrated in Figure 12-4. In the late afternoon, the highest concentrations in this profile were at 900 to 1200 m (3000 to 4000 ft) and adjacent to the mountain slope. The oxidant was not always confined below the inversion layer, but it was present in the inversion layer and above the mountain crest.[18] An instrumented aircraft measured concentrations of oxidant ranging from 100 to 200 $\mu g/m^3$ (0.05 to 0.11 ppm) as high as 2432 m (8000 ft) approximately 1033 m (3400 ft) above the ridge crest during several flights at noon and at 4 p.m. At these times, downwind diffusion of oxidant beyond the crest, on the basis of measurements taken on aircraft flights approximately 150 m (500 ft) above the terrain, suggested only slight dilution in the first 10 km (6 miles) north of the crest.[18] Observations on other days[34] suggested a 50- to 60-percent reduction in oxidant 10 km (6 miles) north of the crest. There was no ground station at this point for comparison with the aircraft measurements at either time.

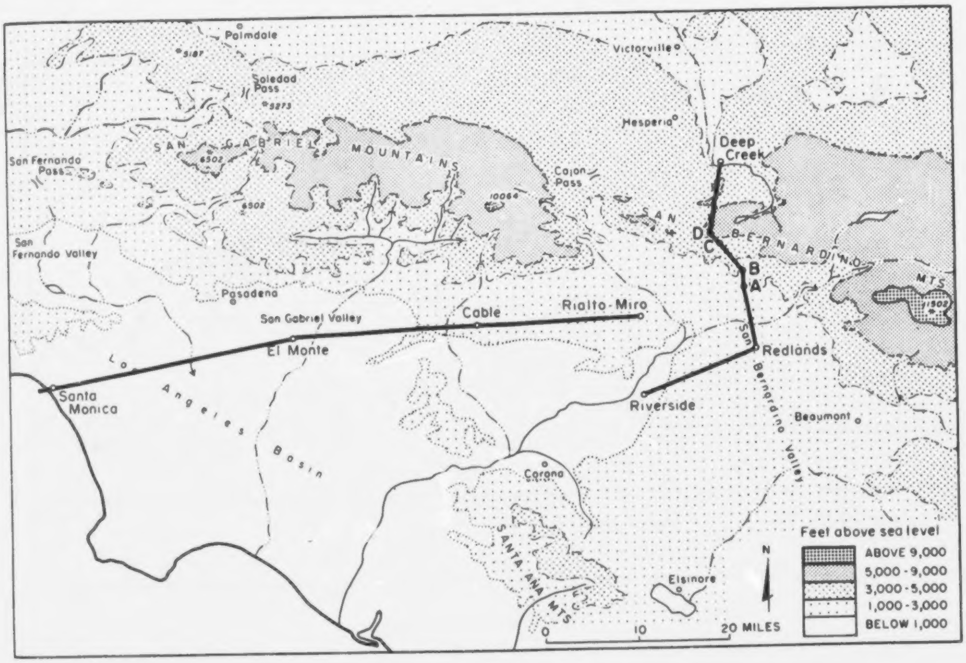
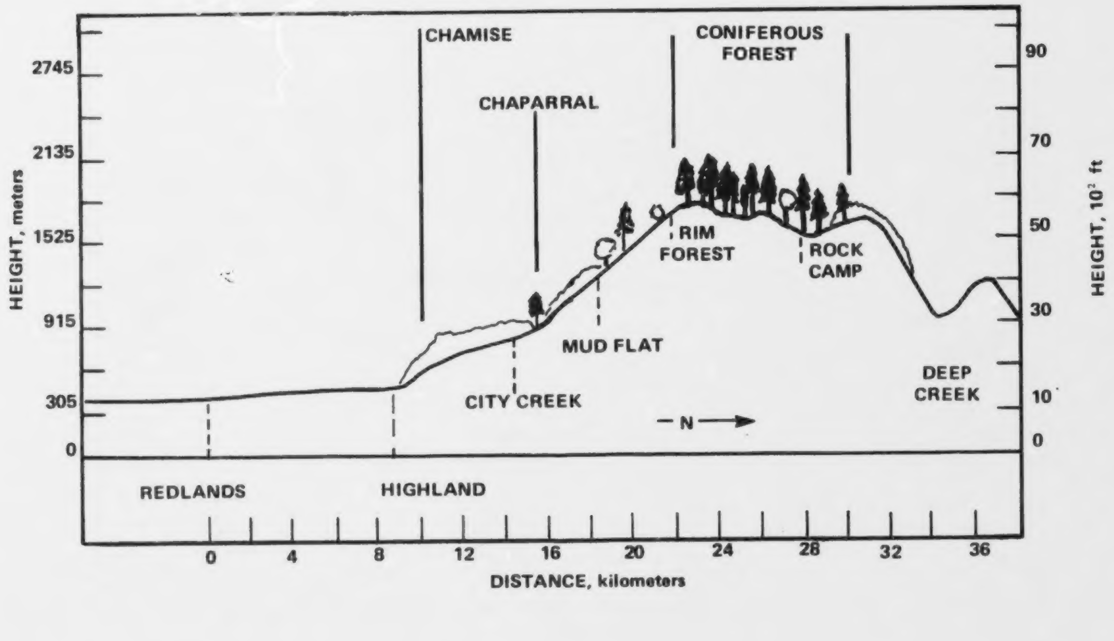

Palmdale
5187
Soledad Pass
5273
Victorville
Hesperia
SAN GABRIEL MOUNTAINS
6502
6502
10064
Cajon Pass
San Fernando Pass
San Fernando Valley
Deep Creek
SAN BERNARDINO MTS
D
C
B
A
11502
Pasadena
San Gabriel Valley
Cable
Rialto-Miro
El Monte
Santa Monica
Los Angeles Basin
San Bernardino Valley
Redlands
Riverside
Beaumont
Corona
SANTA ANA MTS
Elsinore
N
0
10
20 MILES
Feet above sea level
ABOVE 9,000
5,000-9,000
3,000-5,000
1,000-3,000
BELOW 1,000

Figure 12-2. Major topographic features of the Los Angeles Basin with inland valleys and mountains. Station locations: A, Highland; B, City Creek; C, Mud Flat; D, Rim Forest. Aircraft flight paths for the study area are also shown.[18]

Total oxidant, temperature, and vapor-pressure gradient were measured continuously during 16 days in July and August at the mountain slope and crest stations (A, B, C, and D, in Figure 12-2). Figure 12-5 shows that the time of the daily peak oxidant concentration was progressively later at stations of higher elevation. Temperatures and vapor-pressure gradients were also progessively lower at higher elevations at the time of oxidant peak. The duration of oxidant concentrations exceeding 200 $\mu g/m^3$ (0.10 ppm) was 9, 13, 9 and 8 hr/day going from lower to higher stations. The longer duration at City Creek (elevation 817 m, or 2680 ft) probably coincides with the zone where the inversion layer most often contacts the mountain slope. The oxidant concentrations rarely decreased below 98 $\mu g/m^3$ (0.05 ppm) at night on the slope of the mountain crest, whereas they usually decayed to near zero at the basin station (Highland).

The vegetation zones along the slope and at the crest are subjected to oxidant exposure differently. Even though the lower chaparral receives longer exposure, the peak concentrations coincide more closely with the maximal evaporative stress for the day. There is some support for the hypothesis that plant stomata would be closed during this period and that pollutant uptake would thus be lower. There is, in fact, very little visible injury to the species in this zone; however, chaparral is more sclerophyllous and therefore less likely to show visible injury. In contrast, the daily oxidant peak occurs well after the maximum temperature and vapor-pressure gradients have occurred in the conifer forest at the mountain crest, where oxidant injury to plant species is severe. These suggestions of possible microclimatic control of the sensitivity of these native species to ozone form a working hypothesis that needs further investigation.



CHAMISE
CONIFEROUS FOREST
CHAPARRAL
RIM FOREST
ROCK CAMP
MUD FLAT
CITY CREEK
DEEP CREEK
— N →
REDLANDS
HIGHLAND
HEIGHT, meters
2745
2135
1525
915
305
0
HEIGHT, 10^2 ft
90
70
50
30
10
0
0
4
8
12
16
20
24
28
32
36
DISTANCE, kilometers

Figure 12-3. Altitudinal sequence of ecosystems in the San Bernardino Mountains.[34]

San Joaquin Valley and Adjacent Sierra Nevada Mountains

Field surveys[36,69] have confirmed oxidant injury to ponderosa pine and associated species at numerous locations in the Sierra Nevada foothills east and southeast of Fresno. Oxidant measurements at ground stations and by instrumented aircraft show late-afternoon peaks of transported oxidant on the western slopes of the Sierras. Limited measurements by instrumented aircraft suggest the development of a layer of oxidant approaching the forested mountain slopes between 610 and 1829 m (2000 and 6000 ft) during the late afternoon.[35] A very weak inversion or isothermal layer may serve as a reservoir of oxidant, which is advected to the mountain slope in the southern coastal air basin, as suggested by Edinger.[17]

Considerable concern has been registered about air quality in the Lake Tahoe Basin, where local development may cause adverse oxidant concentrations.

Seasonal and Daily Variations of Injurious Concentrations—Synoptic Weather Patterns Associated with Episodes of High Pollution

McCutchan and Schroeder[30] classified 145 days during May through September 1970 with respect to pollution and meteorology on the basis of data collected on the southern slopes of the San Bernardino Mountains. Five general weather patterns were described, along with the associated synoptic patterns at the surface and at 500 millibars (mb) (Table 12-2). An analysis of eight meteorologic variables was used to classify days during May through September into the five general categories. Of 123 days classified by stepwise discriminant analysis, 10, 13, 44, 23, and 24 were placed in classes 1 through 5, respectively; the remaining 9 days could not be

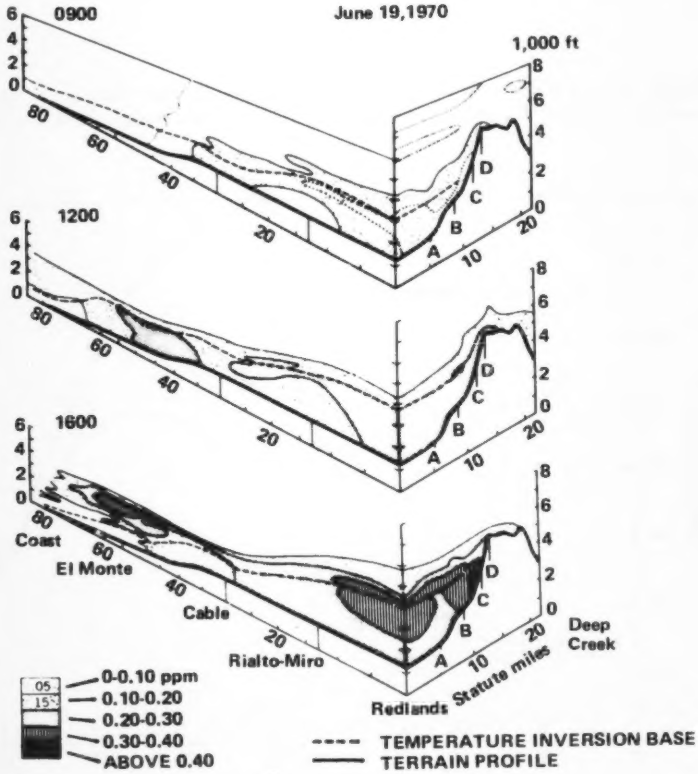


Figure 12-4. Daytime changes in oxidant concentrations along a west-to-east transect in the southern coastal air basin, including the slopes of the San Bernardino Mountains (see Figure 12-2). Reprinted with permission from Edinger et al.[18]

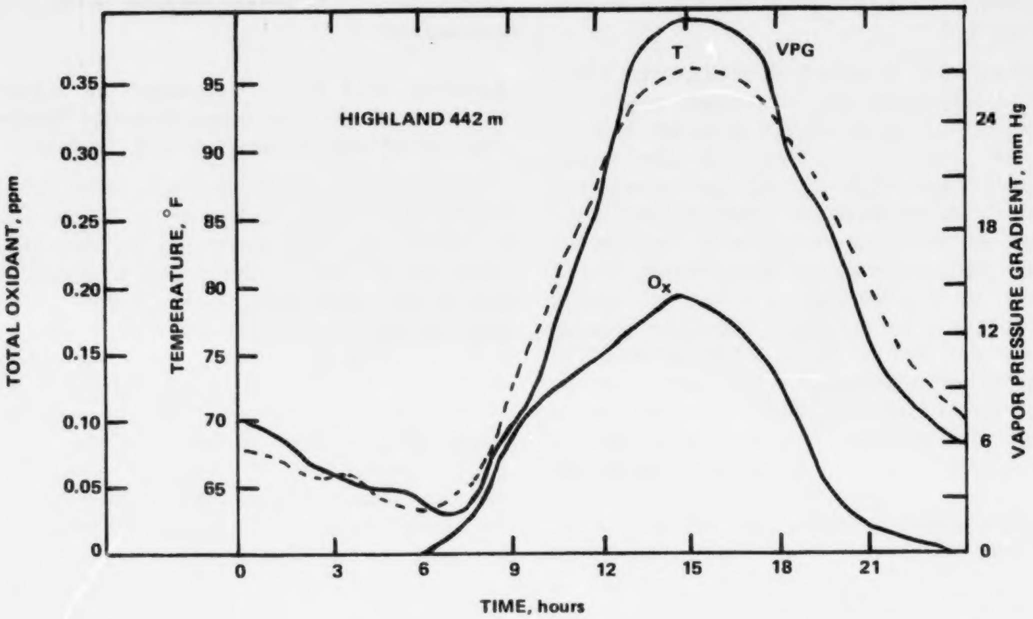


Figure 12-5. The relationship of time of occurrence of the daily peak oxidant concentration to temperature and vapor-pressure gradients in an elevational sequence (see Figures 12-1 and 12-2) on the slopes of the San Bernardino Mountains, July-August 1969.[34]

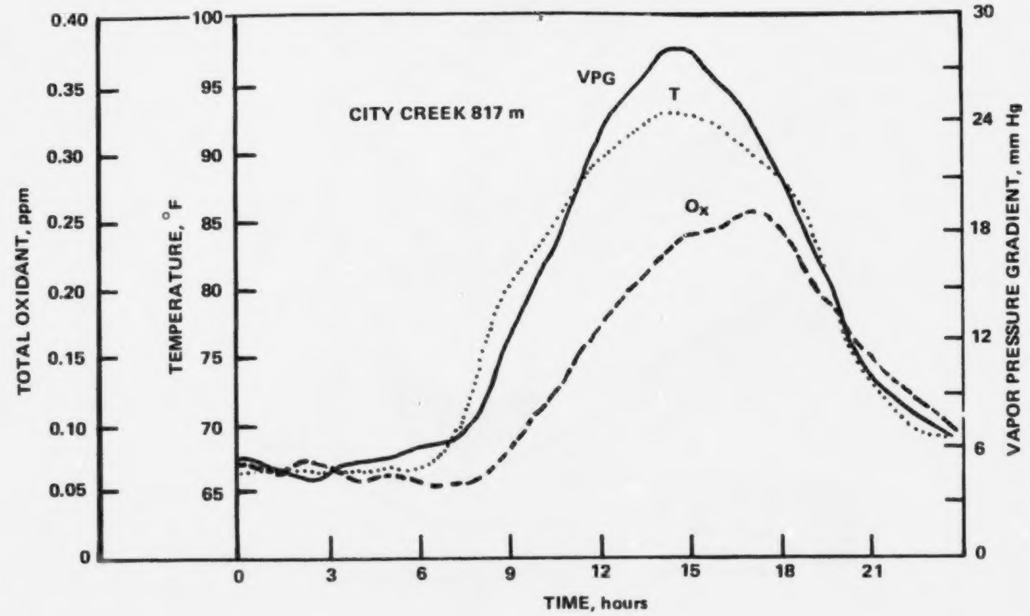


Figure 12-5 (continued). The relationship of time of occurrence of the daily peak oxidant concentration to temperature and vapor-pressure gradients in an elevational sequence (see Figures 12-1 and 12-2) on the slopes of the San Bernardino Mountains, July-August 1969.[34]

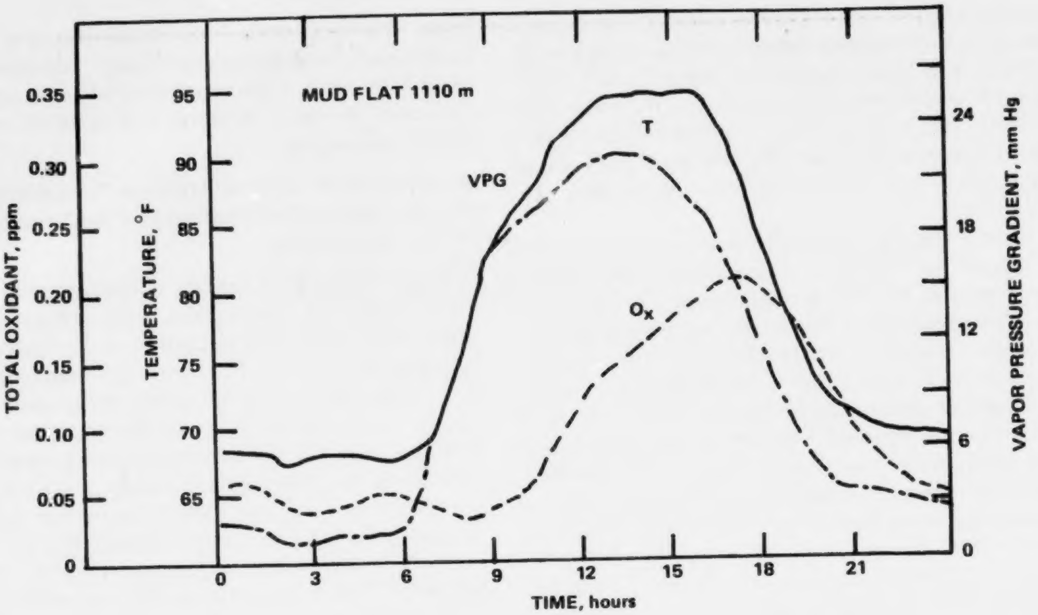


Figure 12-5 (continued). The relationship of time of occurrence of the daily peak oxidant concentration to temperature and vapor-pressure gradients in an elevational sequence (see Figures 12-1 and 12-2) on the slopes of the San Bernardino Mountains, July-August 1969.[34]

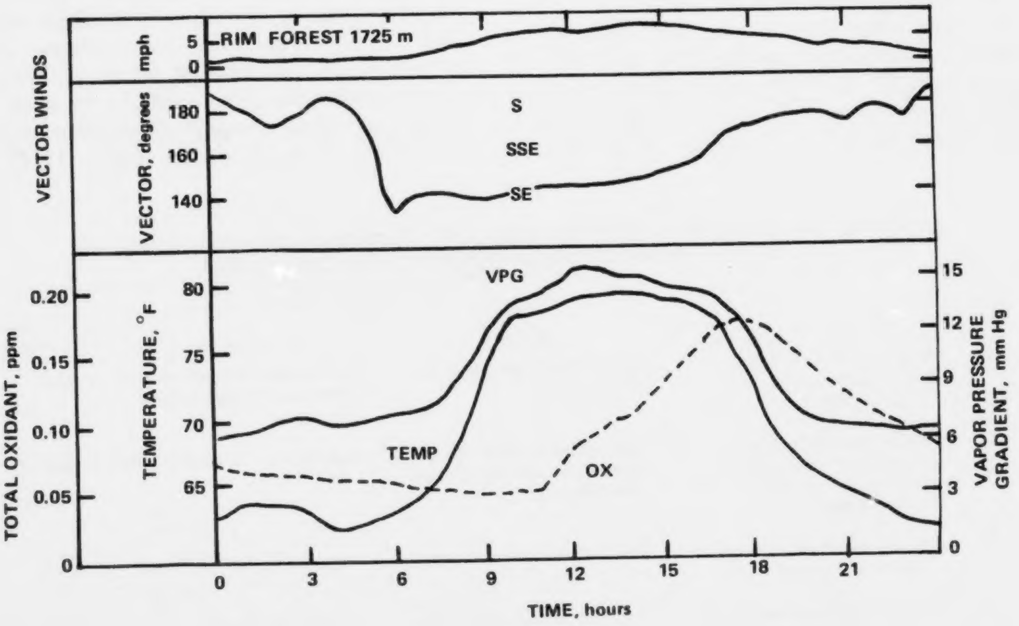


Figure 12-5 (continued). The relationship of time of occurrence of the daily peak oxidant concentration to temperature and vapor-pressure gradients in an elevational sequence (see Figures 12-1 and 12-2) on the slopes of the San Bernardino Mountains, July-August 1969.[34]

placed clearly in any of the five classes. The oxidant concentrations associated with these classes were Class 1, low; Class 2, low and high; Class 3, high; Class 4, moderately high; and Class 5, low. In this sample, the sum of days in classes 2 and 3 (57 days) suggests that about 46 percent of the sample days had high concentrations of oxidant air pollutants.

Class 2 and 3 days are both characterized by high pressure (a 500-mb ridge over the area). Descriptions of the synoptic weather patterns (contained in the U.S. Forest Service *California Fire Weather Severity, 10-Day Summaries*) for May through August 1972-74 were compared with episodes of severe oxidant concentration at Rim Forest and nearby Sky Forest. For the purposes of this comparison, synoptic patterns were reviewed for all days having a maximum hourly concentration of 646 $\mu g/m^3$ (0.33 ppm) or higher. In all cases, the persistent 500-mb high pressure over the area was the most common synoptic feature. For 8 qualifying days in 1972, the mean of the maximum hourly concentrations was 724 $\mu g/m^3$ (0.37 ppm); for 16 days in 1973, it was 803 $\mu g/m^3$ (0.41 ppm); and for 46 days in 1974, it was 744 $\mu g/m^3$ (0.38 ppm). The highest hourly concentration obtained at that time was 1176 $\mu g/m^3$ (0.60 ppm) on June 28, 1974.

During episodes of severe oxidant pollution, the weather is generally very hot (85° to 100°F, or about 29° to 38°C). The relative humidity may be either low or moderately high on class 2 and 3 days, depending on the behavior of the marine layer. The small difference in the means of maximum hourly concentrations on high-pollution days in 1972 through 1974 suggests that heavy primary pollutant emission continues in spite of current control strategies.

Annual Trends of Total Oxidant Concentrations at a San Bernardino Station and the Nearby City of San Bernardino

Since 1968, total oxidant concentrations have been measured continuously with a Mast ozone meter (calibrated by the California Air Resources Board method) from May through September at Rim Forest/Sky Forest.[34] The fall, winter, and early spring months have generally been omitted until recently, because synoptic patterns are usually not conducive to oxidant accumulation and transport. For example, average maximum hourly oxidant concentrations from October through March 1973 and 1974 stayed below 196 $\mu g/m^3$ (0.10 ppm); those for March were 196 to 294 $\mu g/m^3$ (0.10 to 0.15 ppm).[34] The main data collection period coincides with the growing season and thus permits a reasonable estimate of the total annual dose of oxidant air pollutant received by vegetation.

In documentation of oxidant concentration trends during the 1968 to mid-1976 period at Rim Forest/Sky Forest, the accumulated dose is expressed as the sum of all hourly values (in $\mu g/m^3$), including concentrations for each month separately, June through September (the main part of the growing season) (Figure 12-6). These

TABLE 12-2. DESCRIPTIONS OF METEOROLOGIC PATTERNS FOR FIVE CLASSES OF SPRING AND SUMMER DAYS IN SOUTHERN CALIFORNIA[a]

		Associated synoptic pattern		
Class	General weather	At surface	At 500 mb	Oxidant concentration
1	Hot, dry continental air throughout the day (Santa Ana)	Large high pressure over Great Basin	Strong northerly winds over area with trough east of the area	Low
2	Relatively dry forenoon; modified marine air in afternoon; very hot (heat wave)	High pressure over Great Basin and thermal trough over desert	Subtropical closed high over area	Low and high
3	Moist, modified marine air; hot in afternoon	Thermal trough over desert	Ridge over area	High
4	Moist, modified marine air; warm in afternoon	Thermal trough over desert	Trough over area	Moderately high
5	Cool, moist, deep marine air throughout the day	Synoptic low over desert	Deep trough or closed low over area	Low

[a] Modified from Reference 38

doses exclude background concentrations (those less than or equal to 59 $\mu g/m^3$, or 0.03 ppm).[23] The percentage of valid data recovered, as well as the total possible hours for which data could be obtained for each month, June through September, is also indicated. The absence of some data, ranging up to 17.8 percent in 1970, but averaging 8.3 percent during the 7 years, represents a margin of error that cannot at present be adjusted with any certainty. The total number of hours with concentrations of 160 $\mu g/m^3$ (0.08 ppm) or more during June through September was never less than 1300 hours during each of the first 7 years. Recent predictions suggest that oxidant doses will either increase annually or oscillate around the mean of the present high levels unless dramatic improvements occur.[33]

Data from the downtown San Bernardino station operated by the County Air Pollution Control District (APCD) are available back to 1963. The colorimetric potassium iodide method used to measure total oxidants was calibrated according to the method of the California Air Resources Board. A positive correction factor of 1.22 was used to adjust mountain data for the decreased air pressure at the higher elevation.

The data from the Rim Forest/Sky Forest station were compared with published data from the San Bernardino County APCD. The number of hours with concentrations exceeding 392 $\mu g/m^3$ (0.20 ppm) during July, August, and September 1969 through 1974 were compared (Figure 12-7). For 1963 through 1968, data are shown only from the San Bernardino APCD. A large part of the year-to-year differences at the same station and between stations can be attributed to differences in synoptic and mesoscale (local) meteorologic patterns. For example, the increases in 1972, 1973, and 1974 at Rim Forest/Sky Forest are associated with 6, 16, and 46 days, respectively, when a persistent 500-mb ridge occurred over the Southwest, particularly southern California. The difference between stations in the same year is probably influenced most by inversion height. Lower inversions partially restrain transport upslope to shorter periods each day. Higher inversions would have the opposite effect and also allow the greater air volume below to dilute the oxidants. The index for comparison chosen in Figure 12-7 (i.e., hours ≥ 392 $\mu g/m^3$) would be sensitive to inversion height. The 3-year moving averages for each station tend to remove some of the variation resulting from

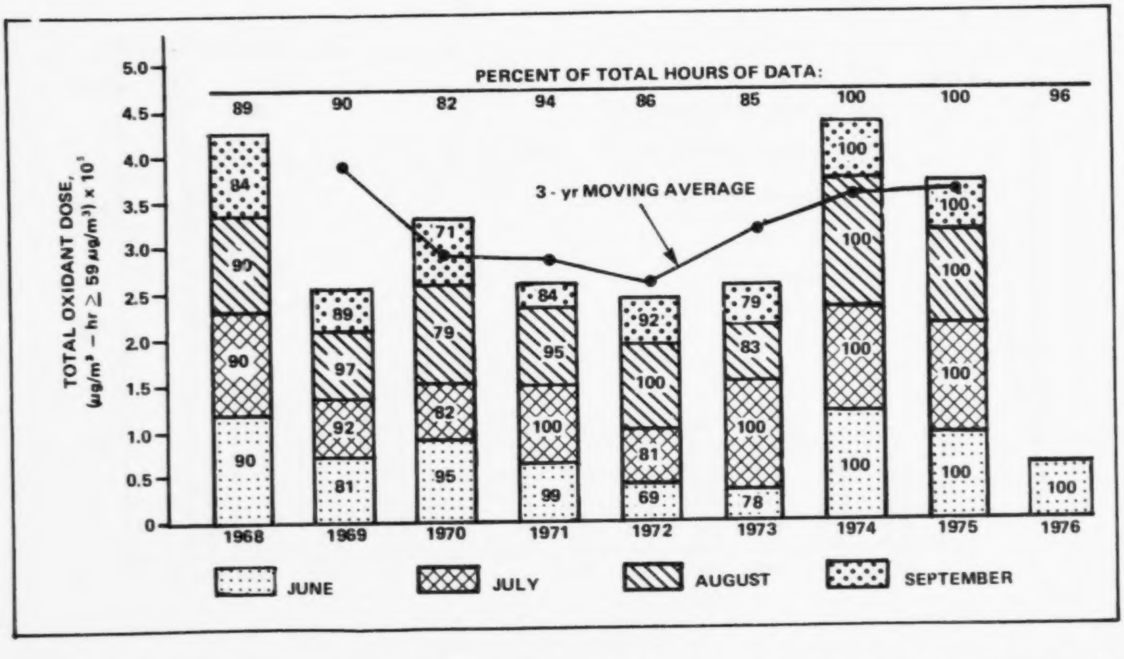


Figure 12-6. Monthly summation of total oxidant concentration data at Rim Forest/Sky Forest, San Bernardino Mountains, California, June through September, 1968-75.[33]

meteorology. In terms of numbers of hours with oxidant equal to or exceeding 392 $\mu g/m^3$, the moving average between 1970 and 1973 at Rim Forest/Sky Forest has increased from 175 to 290 hr. The increased oxidant concentrations at both these stations are the reverse of those in upwind, urban Los Angeles County, where increased emmission of NO_x tends to shift the chemical equilibrium to the left, toward the ozone precursors. The most recent data[13] firmly indicate that oxidant concentrations will either increase annually or continue to oscillate around the mean of present high concentrations in the foreseeable future at these distant locations.

Robinson's tropospheric ozone cycle[13] describing rural upwind, urban, and rural downwind variations in concentration can be easily demonstrated in the Los Angeles and connected inland basins. The effects on both natural ecosystems and agroecosystems also can be demonstrated. For example, oxidant measurements were made during August 1972 at six oxidant stations (Costa Mesa, La Habra, Corona, Riverside, City Creek, and Rim Forest) extending in a line northeastward from the coast to the mountains. These measurements show low doses at the coast, increasing to a maximum on the chaparral-covered mountains and decreasing beyond the mountain crest (Figure 12-8). In Figure 12-8, the oxidant dose is indicated by the dashed line. A crude estimate of the relative economic value of ecosystems encountered along this transect is expressed in a very general way by the solid line and the nomenclature on the abscissa. Finally, the relative complexity of the ecosystems involved is shown below the abscissa. This conceptualization emphasizes the enormously greater dosage received by the natural ecosystems during this month. This pattern of dosages is very typical of June, July, and August, but the offshore flow typified by the Santa Ana winds may reverse the situation in September or October. Susceptible crops growing on the coastal plain may be seriously damaged.

In general, the permanent vegetation constituting natural ecosystems receives much greater chronic exposure than the short-lived, higher-value vegetation constituting the agroecosystems of the coastal plain. The latter can be subjected to injurious doses, but in intermittent, short-term fumigations. Each situation has measurable economic and aesthetic effects, but on different time scales. The simple agroecosystem has little resiliency to pollutant stress; losses are immediate and may be catastrophic. The complex natural ecosystem is initially more resistant to

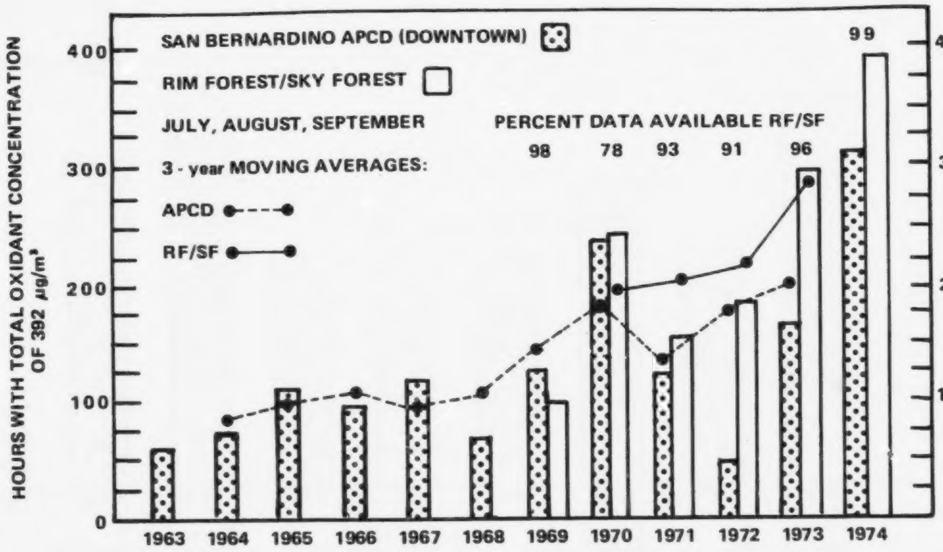


Figure 12-7. Number of hours of total oxidant, July through September, greater than or equal to 392 $\mu g/m^3$ (0.20 ppm) at the downtown San Bernardino County Air Pollution Control District Station, 1963-74, and Rim Forest/Sky Forest, 1968-74.[33]

pollutant stress, but the longer chronic exposures cause the disruption of both structure and function in the system. The damage may not be reversible.

INVESTIGATING THE EFFECTS OF OXIDANT STRESS ON ECOSYSTEMS

Ecosystem Modeling

Ecosystems are very complex and extremely difficult to understand. An approach to understanding these complexities is to describe them in mathematical terms. However, before mathematical shorthand can be used, the components to be studied must be defined and their interactions and relationships with one another identified. To understand the stresses placed on ecosystems by perturbations such as ozone-oxidant air pollution, those elements that could be particularly vulnerable to pollutants must also be identified. The mathematical and computer capability for modeling ecosystems exists, but ecological science does not yet possess sufficient understanding of all of the interactions at the ecosystem level.[2] Therefore, simulation models of forest ecosystems are extremely scarce.[68]

Modeling of the coniferous forest biome is discussed by Overton,[41] and modeling of the eastern forest biome is treated by O'Neill.[40] The usual modeling approach is to select specific processes such as energy flow and nutrient cycling. These processes are a result of the structural interrelationships existing among the various components (soil, water, nutrients, producers, consumers, and decomposers).[48] Energy and nutrients may enter or leave an ecosystem by a variety of pathways. These processes may be described mathematically, and a model may be developed that is an approximation of the real world. This model may then be used to make predictions about the ecosystem. Computer programs for solving the equations explaining interactions of the ecosystem components already exist. Once the model is running smoothly on the computer, it is possible to impose various experimental conditions (e.g., clear cutting or selective cutting) and observe the effects on specific processes such as energy or nutrient flow. Sensitivity analysis can then be performed to determine which transfer coefficients are most sensitive to stress and how much stress is required to cause deterioration of the ecosystem.

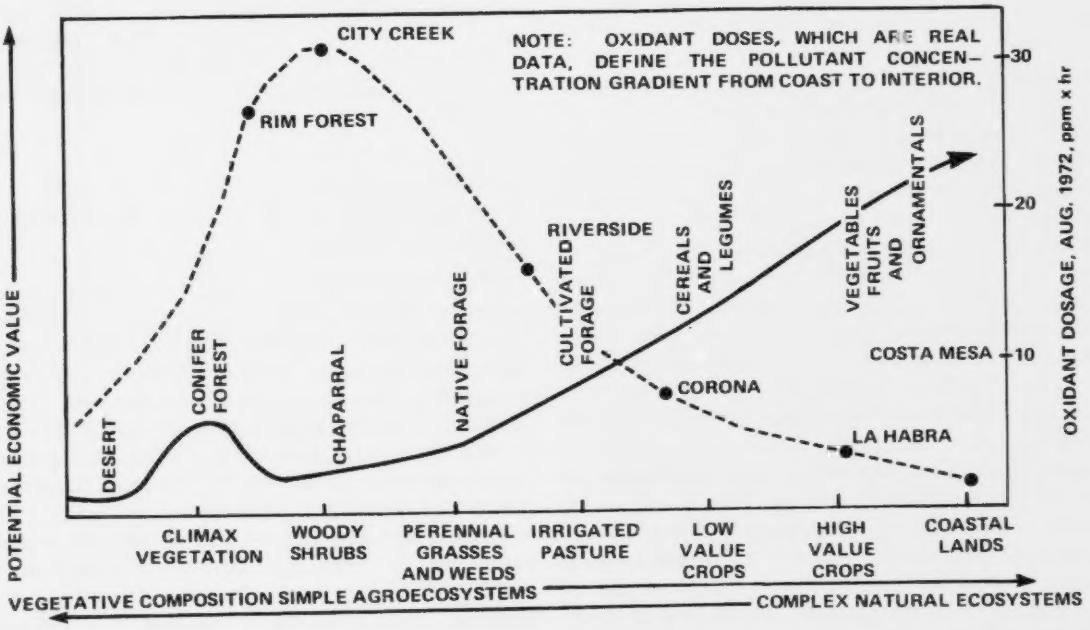


Figure 12-8. Hypothetical relationships of oxidant dose, natural ecosystems, agroecosystems, and the relative values of their vegetation components along a transect of southern coastal air basin of California.[38]

No models dealing with oxidant stress on communities or ecosystems exist; however, models do exist that deal in general terms with disturbance,[29] sensitivity,[8,20,27,44] and stability[8,20,27,44] at community and ecosystem levels. Forest succession has been described by Schugart et al.[47] and Botkin and Miller.[7] The area occupied per biotic unit over time is very important for defining and predicting long-term successional changes in plant communities as a result of pollutant stress. In view of the trend of increasing oxidant concentrations downwind from urban areas,[6] a much more precise understanding of chronic effects on both natural ecosystems and agroecosystems is required. Descriptive and predictive models dealing with the responses of biologic systems can provide the best input to the decision models needed for standard setting, land use planning, and resource management.

Modeling the Effects of Oxidant Stress on a Western Mixed Conifer Forest Ecosystem

The recent history of the mixed conifer forest of the San Bernardino Mountains has been analyzed.[55] The analysis was conducted by nearly a dozen coinvestigators representing many disciplines.[56] This analysis included an initial inventory of ecosystem components and processes, as indicated in Figures 12-9 through 12-13. The inventory emphasizes ponderosa and Jeffrey pines, the most dominant species in the climax community, and is stratified according to organizational level: Organism or tree (Figures 12-9 and 12-10), community or stand (Figure 12-11 and 12-12), and, finally, a time-oriented analysis of plant succession in the aggregation of relatively distinct communities or stands that collectively make up the mixed conifer forest ecosystem (Figure 12-13). These figures present in diagrammatic form the complexity of ecosystem interactions at each level of biological organization. Only the interactions considered to be the most important ones guiding the course of plant succession in the several forest plant communities now constituting the mixed conifer forest have been selected by project investigators for immediate investigation.[57] In the case of each interaction, it is important to decide the time frequency at which the selected variables will be measured (i.e., hourly, weekly, or annually). Because this project is the most active effort to date directed toward understanding the effects of ozone and other oxidants on a natural ecosystem, most of the remainder of this chapter is a discussion of its findings.

Effects on Primary Producers (Green Plants)

The ozone dose responses of both herbaceous and woody plants and the specific effects on the photosynthetic activity (principally in controlled short-term exposures) are discussed in Chapter 11. The aim of this section is to evaluate the effects of the chronic exposure of vegetation in natural ecosystems to total oxidants (more than 90 percent ozone) under field conditions or simulated field conditions. The effects of chronic exposure on agroecosystems are also discussed to a limited extent in Chapter 11.

Smith[51] has suggested three classes of ecosystem response to air pollutants: Those in which vegetation and soils serve only as a sink for pollutants, with no visible vegetation injury; those in which some species, or sensitive individuals within species, are injured and are more subject to other stresses; and those in which high dosages cause acute morbidity or mortality of some species. Although these classes are convenient for discussion, they are not clearly separated in space in most situations. More often, these classes of response occur along a gradient of decreasing pollutant exposure, as in the San Bernardino Mountains.

EXTENT AND INTENSITY OF INJURY TO OVERSTORY TREES IN THE SAN BERNARDINO NATIONAL FOREST

Wert[66] used aerial photography to determine the extent of oxidant injury to ponderosa and Jeffrey pines in diameter classes larger than 30 cm. Injury was categorized as heavy, moderate, light, or negligible, and it generally decreased with distance from the source area. Of the 64,380 ha (160,950 acres) of ponderosa-Jeffrey type within the forest boundaries, 18,492 ha (46,230 acres) had heavy damage, 21,568 ha (53,920 acres) had moderate damage, and 24,320 ha (60,800 acres) had light or negligible damage. An estimated 1,298,000 trees were affected. Of these, 82 percent were moderately affected, 15 percent were severely affected, and 3 percent had died.

The term "ponderosa-Jeffrey type" is a general term that includes a mosaic of five subtypes described by McBride on the basis of species dominance.[38] These subtypes are: Ponderosa pine forest, ponderosa pine/white fir forest, ponderosa pine/Jeffrey pine forest, Jeffrey pine forest, and Jeffrey pine/white fir forest. The injury by oxidant air pollutants is most intense in the Jeffrey pine types. In the field plots of these various types, as a result of oxidant air pollution, the average area covered by shrubs is only 3.8 percent in the ponderosa types, but 26 percent in the Jeffrey pine types.[38]

Later studies[32,55,56] have expressed the amount of injury to ponderosa and Jeffrey pines and associated tree species in permanent study plots as a numerical score. The range of scores is subdivided into seven categories, ranging from dead to no visible symptoms.[32]

In 1974, all tree species at 19 permanent study plots were scored individually by binocular inspection. The data can be obtained from conifers early in the fall, but the most important deciduous species, black oak, was evaluated during the period from August 28 to 31 to prevent confusion of oxidant-injury symptoms with natural autumn senescence of leaves. The injury to black oak on August 31, 1974, at several representative study sites, and the June-August accumulated dose at nearby monitoring stations are shown in relation to the topographic projection of the San Bernardino Mountains in Figure 12-14.[28] Lower scores mean greater injury. The darkened portion of the bar representing oak injury is for leaf

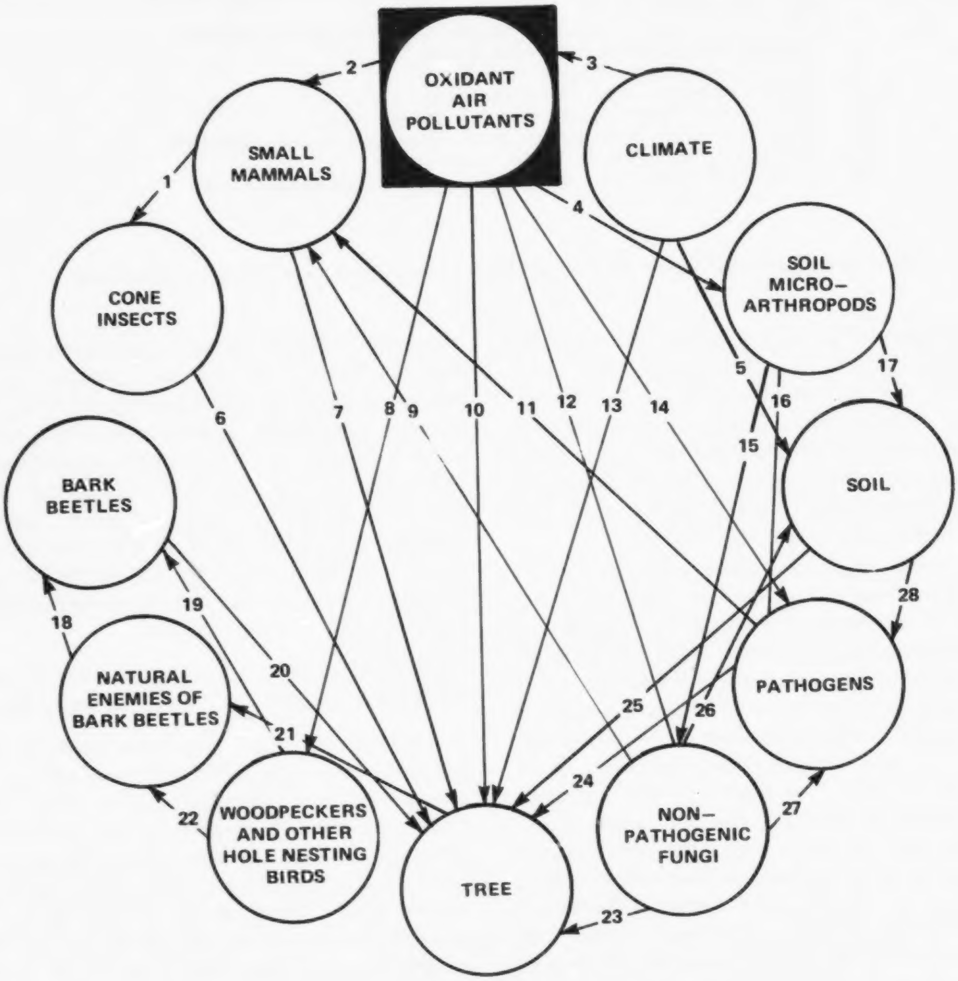

OXIDANT AIR POLLUTANTS
SMALL MAMMALS
CLIMATE
CONE INSECTS
SOIL MICRO-ARTHROPODS
BARK BEETLES
SOIL
NATURAL ENEMIES OF BARK BEETLES
PATHOGENS
WOODPECKERS AND OTHER HOLE NESTING BIRDS
TREE
NON-PATHOGENIC FUNGI
1
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Figure 12-9. Organism-level interactions in a mixed conifer forest.[56]

1. Competition for food supply
2. Direct effect of ozone on small-mammal physiology
3. Climatic control of oxidant concentration in forest
4. Direct effect of ozone in soil microarthropods
5. Effect of precipitation and temperature on soil moisture and soil temperature
6. Insect damage to developing cones
7. Damage by small mammals to developing cones and importance of cone crop as animal food supply
8. Direct effect of ozone on physiology of cavity-nesting birds
9. Role of fruiting bodies of nonpathogenic fungi in nutrition of small animals
10. Direct effect of ozone on tree physiology
11. Role of fruiting bodies of pathogens in nutrition of small animals
12. Direct effect of ozone on nonpathogenic fungi
13. Effect of temperature and evaporative stress on tree growth
14. Direct effect of ozone on pathogenic fungi
15. Interaction of nonpathogenic fungi and soil microarthropods
16. Interaction of pathogenic fungi and soil microarthropods
17. Effect of soil microarthropods on litter reduction
18. Impact of predation and parasitism on bark beetles
19. Predation of bark beetles by woodpeckers
20. Effect of bark beetles on tree mortality and vigor and effect of phloem thickness and moisture on bark beetles
21. Effect of phloem moisture and thickness on natural enemies of bark beetles
22. Influence of woodpeckers on rate of parasitism
23. Effect of nonpathogenic fungi on tree growth
24. Effect of pathogens on tree vigor and mortality
25. Effect of soil moisture and soil temperature on tree growth
26. Effect of soil moisture and temperature on occurrence of nonpathogenic fungi
27. Interaction of pathogenic and nonpathogenic fungi
28. Effect of soil moisture and temperature on occurrence of pathogenic fungi

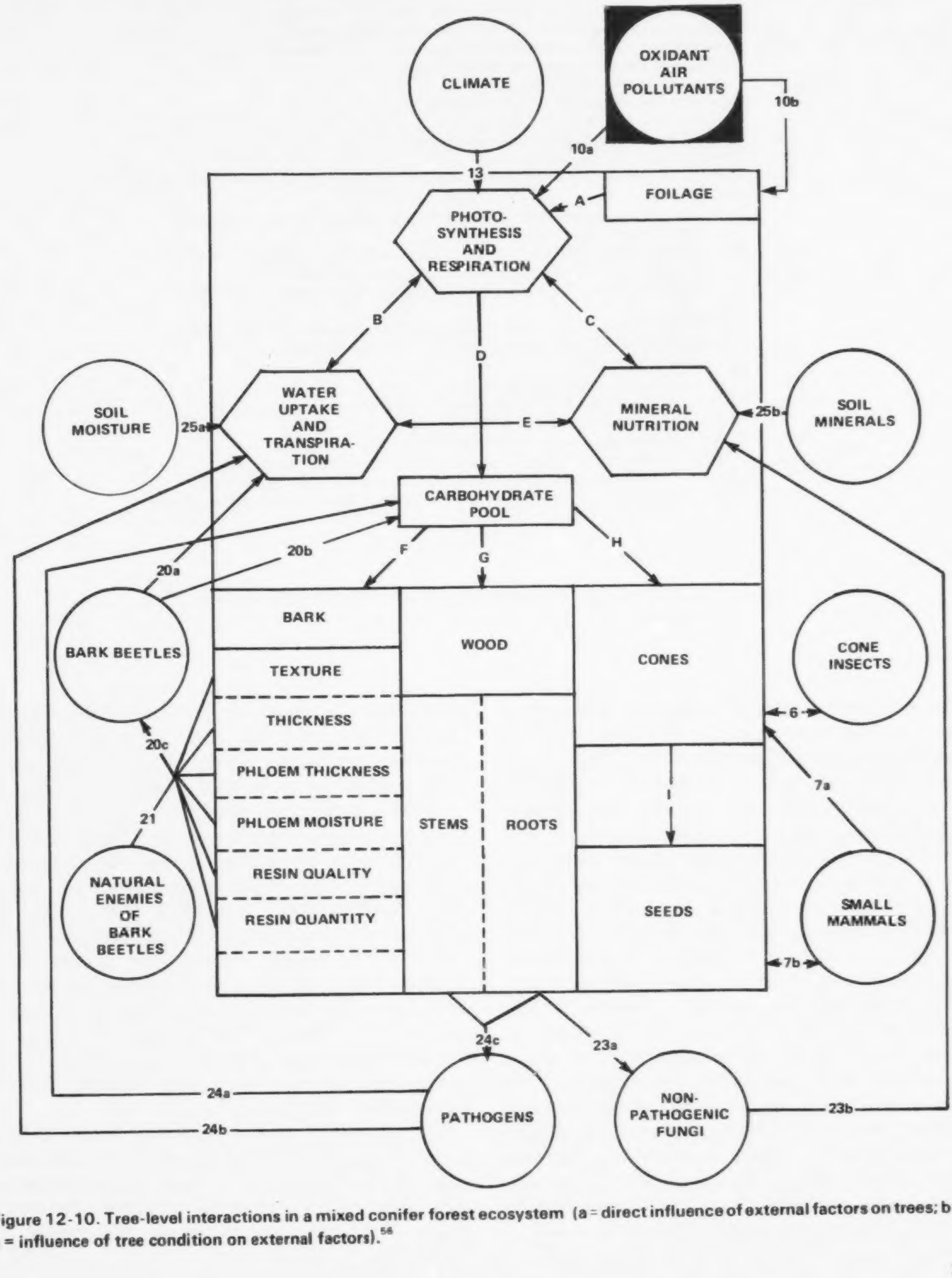
Figure 12-9 (continued). Organism-level interactions in a mixed conifer forest.[56] Types of interactions.

chlorotic mottle and interveinal necrosis. A score of 8 means no injury. The remaining portion of the score is the sum of scores for leaf complement, leaf size, and twig mortality (not shown separately). These data suggest that oak shows no visible injury where the accumulated June-August dose does not exceed about $2.5 \times 10^5 \mu g/m^3$ times hours from about Snow Valley eastward.

The distribution of ponderosa and Jeffrey pines into various injury classes with respect to the distance of the study site along the gradient of oxidant dose (June-September) is illustrated above the topographic projection in Figure 12-15.[28] It is important to realize that the 1974 distribution into injury classes is also a product of earlier years, when the oxidant concentrations were not always as high as in 1974 (see Figure 12-6). The trend toward greater numbers in the very slight injury (29 to 35) and no visible injury (36 and greater) categories is quite evident in the eastern plots receiving lower doses—for example, Holcomb Valley. The assumption has been made that ponderosa and Jeffrey pine respond similarly to oxidant. Ponderosa pine is replaced by Jeffrey pine in the natural stands east of Camp O'ongo, and they intermix at Barton Flats. The validity of this assumption can be partially verified by examining the distributions of the two species into injury classes at Barton Flats (Figure 12-15).[28] These data indicate reasonable similarity at a common site; but the influence of other environmental variables that change continuously along the oxidant gradient (such as soil moisture availablity, temperature, and humidity) must be examined more intensively to understand the degree to which they influence oxidant susceptibility (see Figures 12-9 through12-13).

OXIDANT DAMAGE TO CONIFER SPECIES IN THE 19 MAJOR STUDY PLOTS FROM 1973 TO 1974

The first evaluation of oxidant injury to all tree species in the new study plots was completed in September and October 1973. The second evaluation in 1974 offered the first opportunity to compare increases in tree injury and mortality. In Table 12-3, the plots are arranged in the order of severe injury (first) to no visible injury (last), according to the 1974 average injury score for all ponderosa or Jeffrey pines in each plot. Of the 12 plots categorized as severe and moderate, all but 6 showed injury significantly increased (lower scores), with a probability of 0.05. Among the six remaining plots where injury was classified as slight, very slight, or not visible, there were three significant increases, one insignificant increase, and two decreases (one significant and one insignificant), all at a probability of 0.05.



CLIMATE
OXIDANT AIR POLLUTANTS
10a
10b
13
FOILAGE
A
PHOTO-SYNTHESIS AND RESPIRATION
B
C
D
SOIL MOISTURE
25a
WATER UPTAKE AND TRANSPIRA-TION
E
MINERAL NUTRITION
25b
SOIL MINERALS
CARBOHYDRATE POOL
20a
20b
F
G
H
BARK BEETLES
BARK
TEXTURE
THICKNESS
PHLOEM THICKNESS
PHLOEM MOISTURE
RESIN QUALITY
RESIN QUANTITY
WOOD
STEMS
ROOTS
CONES
SEEDS
CONE INSECTS
6
7a
7b
SMALL MAMMALS
20c
21
NATURAL ENEMIES OF BARK BEETLES
24c
23a
24a
24b
PATHOGENS
NON-PATHOGENIC FUNGI
23b

Figure 12-10. Tree-level interactions in a mixed conifer forest ecosystem (a = direct influence of external factors on trees; b and c = influence of tree condition on external factors).[56]

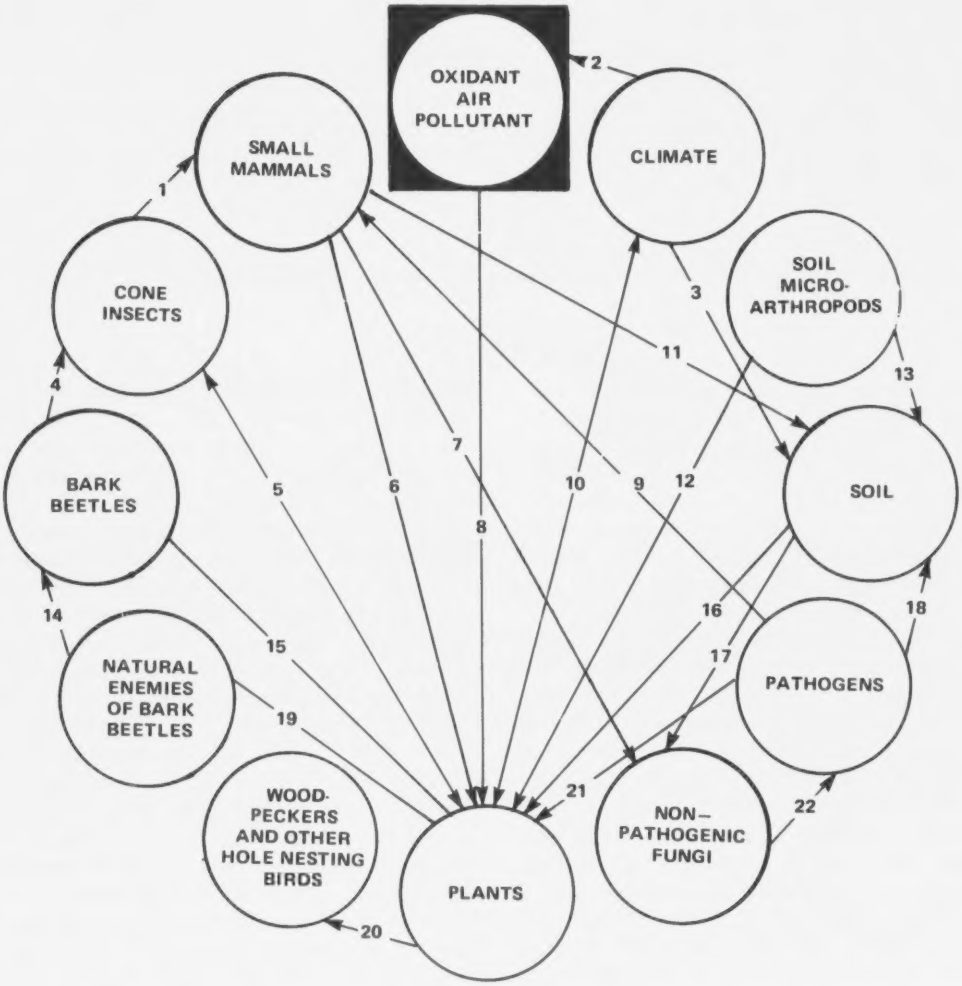
A.	Relationship between net photosynthesis, foliage age, and foliage retention
B.	Effect of plant moisture stress on net photosynthesis
C.	Relationship between mineral uptake and net photosynthesis
D.	Relationship between net photosynthesis and carbohydrate storage
E.	Relationship between water uptake and mineral nutrition
F.	Relationship between carbohydrate storage and bark characteristics
G.	Relationship between carbohydrate storage and wood production
H.	Relationship between carbohydrate storage and cone production
I.	Relationship between cone crop and seed crop
6.	Cone insect damage to developing cones
7a.	Small-mammal damage to developing cones
7b.	Small-mammal predation of seeds
10a.	Effect of ozone on photosynthesis and respiration
10b.	Effect of ozone on needle retention
13.	Effect of temperature, light intensity, and evaporative stress on photosynthesis and respiration
20a.	Effect of bark beetles on water uptake and transpiration, and relationships of tree moisture stress to bark beetle attack
20b.	Effect of bark beetles on tree carbohydrate concentration and relationships of carbohydrate concentration to bark beetle population in trees
20c.	Relationship between bark beetle characteristics and bark beetle attack and population
21.	Relationship between bark characteristics and natural enemies of bark beetles
23a.	Relationship between root characteristics and mycorrhizal-forming non-pathogenic fungi
23b.	Effect of nonpathogenic fungi on mineral uptake
24a.	Effect of pathogens on tree carbohydrate concentration and relationship of carbohydrate concentrations to pathogen attack
24b.	Effect of pathogens on water uptake and transpiration
24c.	Relationship between stem and root characteristics and pathogen attack
25a.	Effect of soil moisture and temperature on water uptake and transpiration
25b.	Effect of soil mineral concentration and temperature on mineral nutrition of tree

Figure 12-10. Tree-level interactions in a mixed-conifer forest ecosystem.[56]

Tree mortality among ponderosa and Jeffrey pines was about the same in 1973 and 1974. The greatest mortality was in the permanent study plots listed as receiving moderate injury. Possibly the populations in these plots still retain greater numbers of the more susceptible genotypes. In previous years, tree mortality rates for ponderosa or Jeffrey pines in the stands suffering moderate to severe injury were 8 and 10 percent from 1968 to 1972,[55] 8 percent from 1969 to 1971,[32] and 24 percent from 1966 to 1969.[12] The death of weakened trees is usually due to the pine bark beetle.[53] Mortality has not been observed in tree species other than ponderosa and Jeffrey pine.

Two 2-ha (5-acre) control plots in the vicinity of Barton Flats in the San Bernardino National Forest provide data for the longest observed period of tree decline, 1952 to 1972.[65] These plots are located in the Jeffrey pines, where trees with a diameter breast height (dbh) of 30.5 cm (12 in.) or greater were measured and their vigor described by judging the risk or probability that they would be susceptible to attack and kill by bark beetles *(Dendroctonus* sp). Risk classes 1 and 2 indicate low-risk trees that would definitely be preserved if trees were being marked for a timber sale. Classes 3 and 4 are high-risk trees that would be marked for removal in a timber sale. In Table 12-4, the changes in merchantable volume in board feet (bd ft) in all four classes are recorded for two control plots in 1952, 1963, and 1972. The increase in volume of trees in the high-risk category since 1952 is astounding, whereas the decreases in volume of low-risk trees and total plot volume are very large. The decrease in total volume is due to one-by-one removal of bark-beetle-killed trees in the plots as indicated by the increase in number of snags and stumps; it is also possibly due to suppressed radial growth.

Information available from the study of the ponderosa pine subtype suggests that the biomass production of overstory species is diminished in proportion to the oxidant dose received. Parmeter et al.[43] observed decreases in the height of ponderosa pine that showed injury symptoms. Injured trees did not respond with greater growth during years with more favorable soil moisture content; but uninjured trees (often side by side with injured trees) did increase in height during these years. In another study[38] of two populations (one ranging in age from 52 to 71 years and the other from 20 to 39 years) of dominant ponderosa pine stands, the influence of the Los Angeles smog was noted. The width of growth rings before 1940 was compared with those after 1940. After the influence of precipitation on growth was evaluated, a difference of 0.20 cm in average annual growth was attributable to oxidant air pollution injury.


OXIDANT AIR POLLUTANT
SMALL MAMMALS
CLIMATE
CONE INSECTS
SOIL MICRO-ARTHROPODS
BARK BEETLES
SOIL
NATURAL ENEMIES OF BARK BEETLES
PATHOGENS
WOOD-PECKERS AND OTHER HOLE NESTING BIRDS
PLANTS
NON-PATHOGENIC FUNGI
1
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22

Figure 12-11. Community-level interactions in a mixed-conifer forest ecosystem.[56]

1. Competition between woodpeckers and small mammals
2. Climate control of oxidant concentration in different forest communities
3. Effect of precipitation and temperature on soil moisture and soil temperature in different forest communities
4. Predation of bark beetles by woodpeckers in different forest communities
5. Effect of cone crop abundance on cone insect populations in different forest communities
6. Effect of cone crop abundance on small-mammal populations in different forest communities
7. Fruiting bodies of nonpathogenic fungi as food for small mammals in different forest communities
8. Smog-caused mortality and morbidity in different forest communities
9. Fruiting bodies of pathogens as food for small mammals in different forest communities
10. Effect of temperature and evaporative stress on species composition in different forest communities
11. Relationship between soil characteristics and population density of burrowing small mammals in different communities
12. Relationship between plants and microarthropod population
13. Relationship between soil characteristics and microarthropod population
14. Bark beetle mortality caused by natural enemies in different forest communities
15. Effect of bark beetles on tree mortality and vigor in different forest communities
16. Relationship between soil characteristics and forest community composition and growth
17. Relationship between soil characteristics and species distribution and behavior of nonpathogenic fungi
18. Relationship between soil characteristics and species distribution and behavior of pathogens
19. Influence of forest community type on populations of natural enemies of bark beetles
20. Woodpecker distribution and density in different forest communities
21. Effect of pathogens on tree vigor and mortality in different forest communities
22. Relationship between nonpathogenic fungi and forest community composition and growth

Figure 12-11. Community-level interactions in a mixed- conifer forest ecosystem.[56]

The growth suppression of ponderosa pine saplings has been demonstrated experimentally by Thompson.[58] Trees grown in a plastic-covered greenhouse and provided with charcoal-filtered air (after an initial lag in the growth of shoots and branches in the first years of the experiment) showed dramatically increased growth when compared to trees grown in ambient air outside or trees grown in a greenhouse with unfiltered air (Figure 12-16).[28] Understory ponderosa pines appear to be more susceptible to oxidant than larger-sized trees.[12] The probable effect of tree mortality on stand composition, based on the present species and size-class composition, is shown in Table 12-5. The incense cedar and sugar pine are the most tolerant to oxidant; however, the sugar pine is present only in very low numbers. The accelerated mortality of ponderosa pines has particular significance in this ecosystem because it is the dominant member of the climax community.

The direct effects of ozone on plant species constituting the shrub layer in the conifer forest are not yet sufficiently understood to permit any conclusions to be drawn. Initial field observations suggest that at least 10 species are obviously injured by total oxidant in areas receiving high dosages. These areas have been subject to exposure for at least 20 years, so some species or subspecies of annual plants may have been completely eliminated.

In a series of experiments, Treshow and Stewart[64] fumigated native plants growing under natural field conditions. The ozone concentrations required to injure the more prevalent species in the grassland-oak and aspen-conifer communities during 2-hr exposures to 290, 490, 588, and 785 $\mu g/m^3$ (0.15, 0.25, 0.30, and 0.40 ppm) ozone were determined. Seventy common plant species indigenous to the plant communities were fumigated. Of these, 5 species were injured at 290 $\mu g/m^3$ (0.15 ppm), and 20 species at 490 $\mu g/m^3$ (0.25 ppm). These species included grasses, perennial forbs, and trees, and they were distributed over all vegetation zones (see Table 12-6). The sensitivity of aspen in the aspen-conifer community was considered of great significance. Not only is it dominant, but its presence is vital to the integrity of the community. The shade of the aspen is required by the seedlings of the white fir *(Abies concolor)* and some other species of plants for growth. In the aspen forest of the Wasatch Mountains, where this study was conducted,[64] the tree canopy acted as an effective filter. When ozone concentrations of 1770 $\mu g/m^3$ (0.9 ppm) were measured in the canopy and in clearings, the concentrations under the canopy were only 196 μg/m³ (0.1 ppm). It was also noted that though one exposure to 290 μg/m³ (0.15 ppm) appeared to produce no effect, repeated exposures could injure both growth and reproduction. The implications for possible imbalances in community stability are readily apparent if these plant communities received dosages similar to those typical in the San Bernardino Mountains.

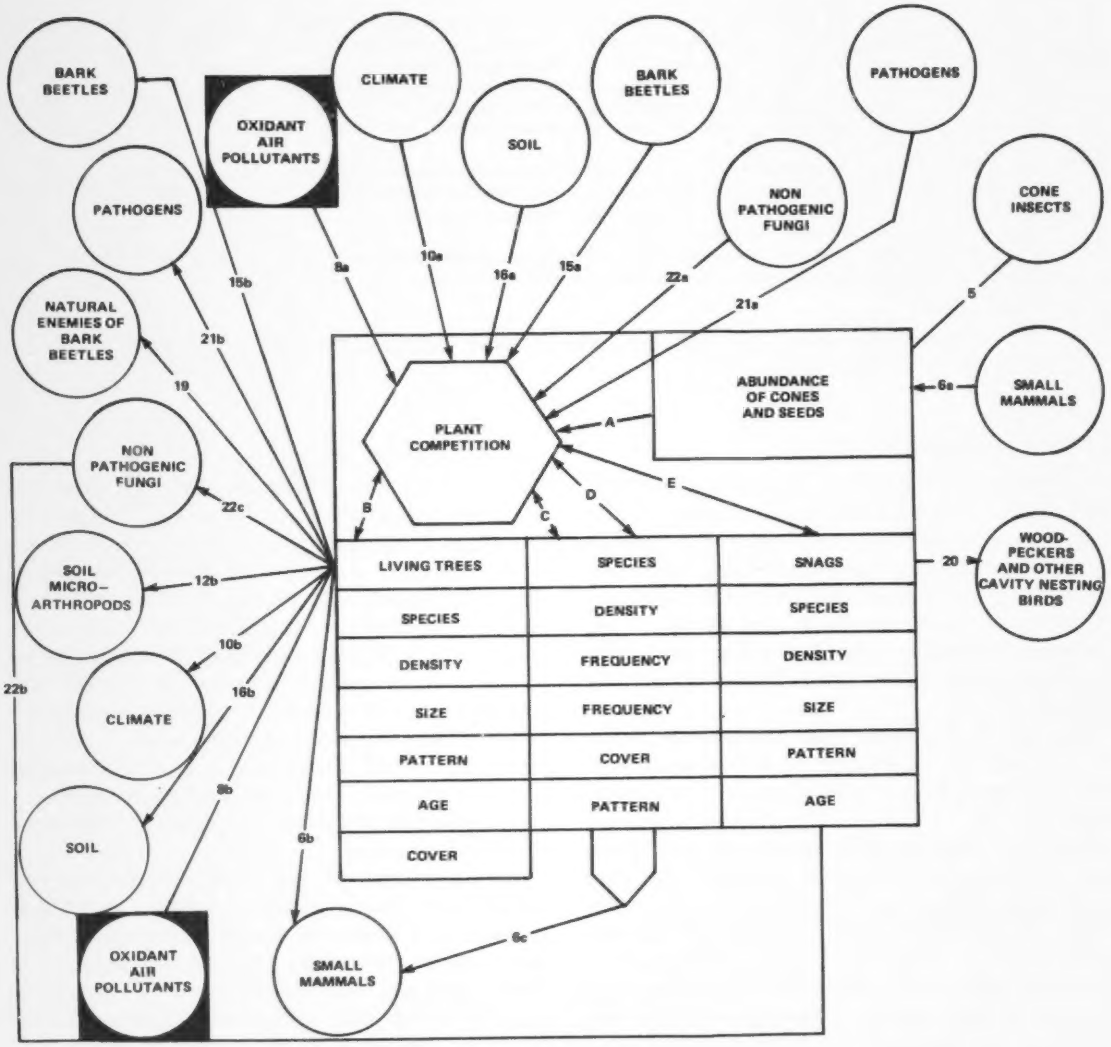

BARK BEETLES
OXIDANT AIR POLLUTANTS
CLIMATE
SOIL
BARK BEETLES
PATHOGENS
NON PATHOGENIC FUNGI
CONE INSECTS
PATHOGENS
NATURAL ENEMIES OF BARK BEETLES
15b
8a
10a
16a
15a
22a
21a
5
21b
19
ABUNDANCE OF CONES AND SEEDS
6a
SMALL MAMMALS
PLANT COMPETITION
A
NON PATHOGENIC FUNGI
B
C
D
E
22c
LIVING TREES
SPECIES
DENSITY
SIZE
PATTERN
AGE
COVER
SPECIES
DENSITY
FREQUENCY
FREQUENCY
COVER
PATTERN
SNAGS
SPECIES
DENSITY
SIZE
PATTERN
AGE
20
WOOD-PECKERS AND OTHER CAVITY NESTING BIRDS
SOIL MICRO–ARTHROPODS
12b
10b
22b
16b
CLIMATE
8b
6b
SOIL
6c
OXIDANT AIR POLLUTANTS
SMALL MAMMALS

Figure 12-12. Stand-level interactions in a mixed conifer forest ecosystem (a = direct influence of external factors on trees in stand; b and c = influence of stand condition on nonpathogenic fungi)[56]

A. Effect of plant competition on abundance of cone and seeds
B. Effect of plant competition on characteristics of living trees
C. Effect of plant competition on characteristics of shrubs
D. Effect of plant competition on characteristics of herbs
E. Effect of plant competition on tree mortality

5. Abundance of cones and seeds and predation by cone insects
6a. Abundance of cones and small-mammal predation of cones
6b. Characteristics of forest stands and small-mammal populations
6c. Influence of shrub and herb layer vegetation on small-mammal populations
8a. Smog-caused mortality and morbidity in different tree species with forest stands
8b. Influence of stand conditions on concentration of oxidants
10a. Influence of temperature and evaporative stress on stand structure and composition
12b. Influence of stand condition on temperature and evaporative stress
15a. Influence of bark-beetle caused tree mortality on stand condition
15b. Influence of stand condition on bark beetle population
16a. Influence of soil moisture and temperature on stand characteristics
16b. Influence of stand condition on soil characteristics
19. Influence of stand condition on population dynamics of natural enemies of bark beetles
20. Relationship between smog occurrence and woodpecker population
21a. Influence of pathogen-caused mortality on stand condition
21b. Influence of stand condition on pathogen population
22a. Influence of mycorrhiza fungi
22b. Influence of snags (and downed trees) on nonpathogenic fungi
22c. Influence of stand condition on nonpathogenic fungi

Figure 12-12 (continued). Stand-level interactions in a mixed conifer forest ecosystem.[56]

EFFECTS ON REPRODUCTION

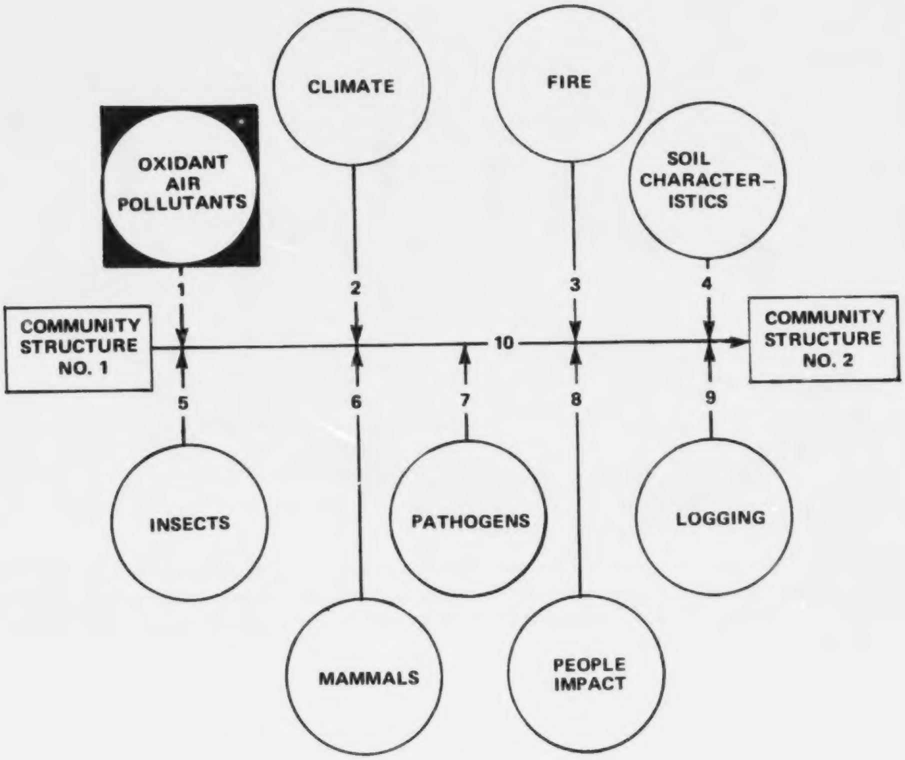
The effect of ozone injury on herbaceous plant reproduction has been mentioned earlier in this chapter and in Chapter 11. Seed production by annuals is influenced mainly by the environmental conditions of the current year, but perennial woody plants, particularly conifers, are erratic seed producers. Factors affecting cone production include age, vigor, seasonal temperature, and soil moisture.[21]

Decrease in tree vigor may decrease or totally eliminate cone production or result in a decrease in the size, weight, and germination of the seed produced.[45,46]

Fruit and seeds make up the largest part of the diet of most of the common small mammals on the study sites, particularly the deer mouse *(Peromyscus* sp.), harvest mouse *(Reithrodontomys)*, chipmunk *(Eutamias)*, ground squirrel *(Callospermophilus)*, and western gray squirrel *(Sciurus griseus anthonyi)*. The gray squirrel is an excellent example of the interactions within this forest and of the potential effects of oxidant air pollution. It is abundant throughout the mixed-conifer-type forest, depending specifically on the pines and oaks for most of its food, cover, and nest sites. This squirrel eats or stores a major portion of the pine and oak seed crops each year. On some yellow pine trees in the study plots, gray squirrels cut more than 2000 cones per tree. During periods of low seed production because of diminished tree vigor, squirrels converge on the few remaining vigorous ponderosa pines and consume about half the seed crop before it matures and reaches the ground. In the areas severely affected by ozone (oxidant), squirrels return to the same trees year after year. After the seed reaches the ground, other small vertebrates, such as mice, seek it out. The habit of preferential seed use by small vertebrates, when considered additively with ozone injury, could seriously reduce the regeneration potential of ponderosa pine.

Thus tree squirrels are a major souce of loss of seeds from the ponderosa, Jeffrey, and sugar pines, and of black oak acorns. Vertebrates, then, can have a major effect on the reproduction of these species, particularly because the gray squirrel is only one of numerous species in this forest that feed on conifer seeds and acorns.

Removal of the dominant species in a specific community can have a profound effect on reproduction of other species. Mention has already been made of the effect of the disappearance of the aspens from the aspen-conifer communities. The


CLIMATE
FIRE
OXIDANT AIR POLLUTANTS
SOIL CHARACTER-ISTICS
1
2
3
4
COMMUNITY STRUCTURE NO. 1
10
COMMUNITY STRUCTURE NO. 2
5
6
7
8
9
INSECTS
PATHOGENS
LOGGING
MAMMALS
PEOPLE IMPACT

1. Oxidant modification of community structure
2. Climate modification of community structure
3. Fire modification of community structure
4. Soil characteristics and their influence on community structure
5. Insect modification of community structure
6. Mammal modification of community structure
7. Pathogen modification of community structure
8. People impact on community structure
9. Logging modification of community structure
10. Predictive capability through integration of submodels for items 1-9

Figure 12-13. Community-succession interactions in a mixed conifer forest ecosystem.[56]

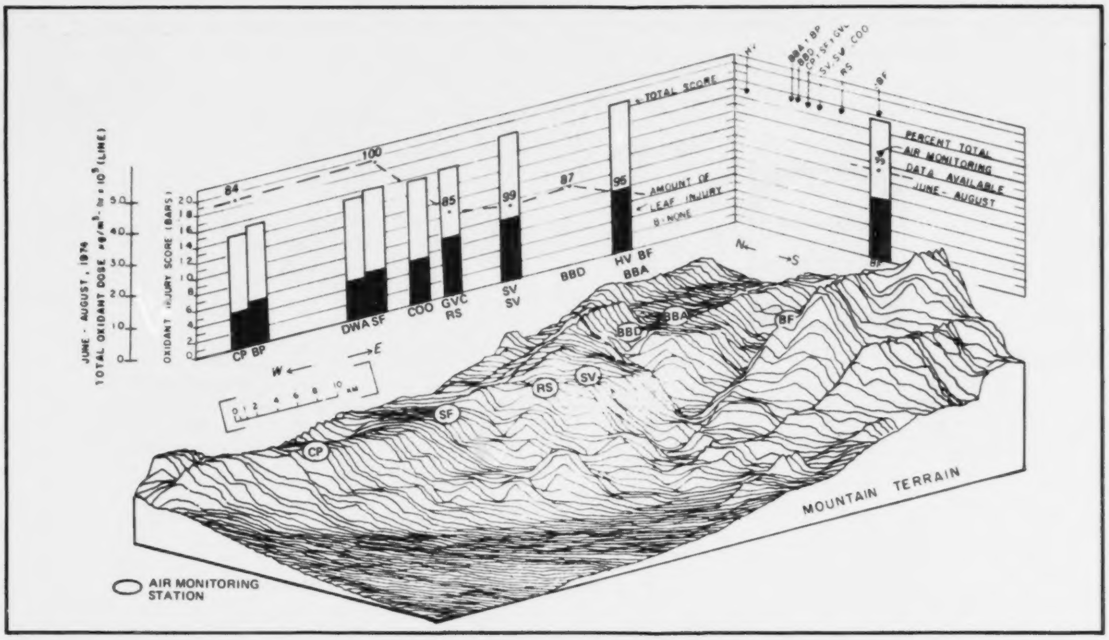
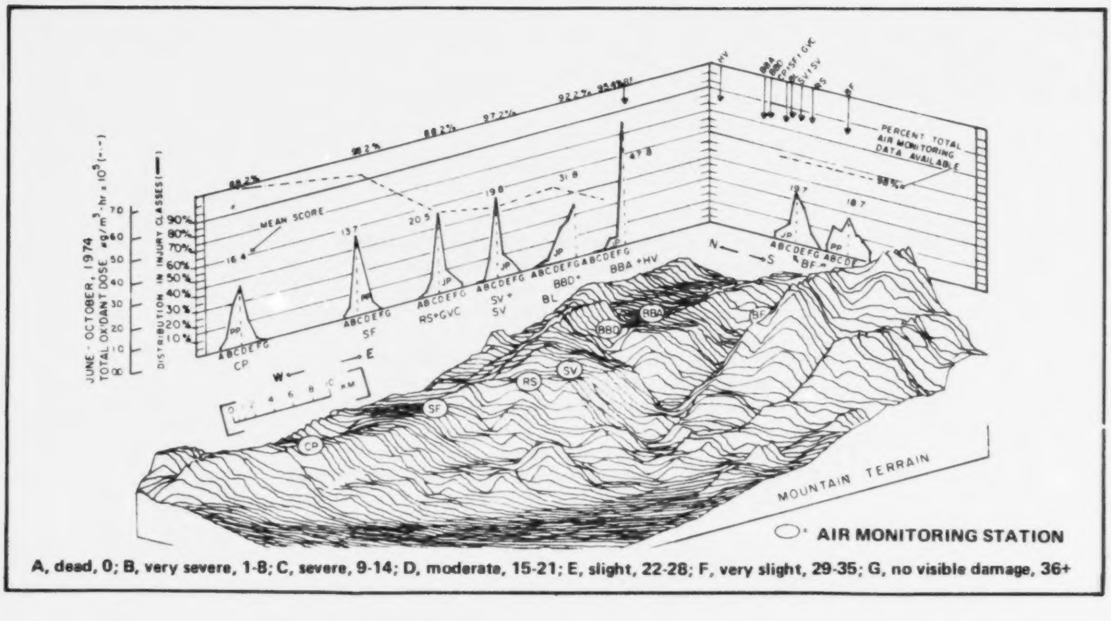


Figure 12-14. Topographic projection, San Bernardino Mountains, with comparison of oxidant injury to black oaks at major study sites, August 31, 1974, with accumulated total oxidant dose for June-August measured at nearby monitoring stations.[28]



A, dead, 0; B, very severe, 1-8; C, severe, 9-14; D, moderate, 15-21; E, slight, 22-28; F, very slight, 29-35; G, no visible damage, 36+

Figure 12-15. Topographic projection, San Bernardino Mountains, showing how ponderosa pines (PP) and Jeffrey pines (JP) in major study sites are distributed in six injury classes according to seasonal dose of total oxidant.[28]

reproduction of white fir is reduced or eliminated, since the shade required by white fir seedlings is no longer present.[64] Other shade-tolerant species are also affected.

Changes in such physical factors as light, temperature (particularly maximums and minimums), relative humidity, and wind speed for forest communities subject to structural alteration by mortality of susceptible species could change the suitability of some sites for growth, reproduction, and reestablishment of survivor species. Some of the possible secondary effects are only speculative until more data are gathered.[32]

EFFECTS ON CONSUMER POPULATIONS

Vertebrate Populations

The effects of oxidant air pollutants on vertebrates can be separated into direct and indirect categories. Direct effects include clinical and pathological alteration of tissues as a result of

TABLE 12-3. CHANGES IN OXIDANT INJURY SCORES AND MORTALITY RATES OF PONDEROSA AND JEFFREY PINES AT 18 MAJOR STUDY PLOTS, 1973-74[38]

Plot	Species	Tree density,[a] 1973	Average injury score[b] 1973	Average injury score[b] 1974	Mortality rate, % 1973	Mortality rate, % 1974	Accumulated mortality, % 1974	Injury description
Schnelder Creek	JP	28	12.4	11.7	0.0	0.0	0.0	Severe
Camp O'ongo	PP	90	15.1	12.9[b]	0.0	0.0	0.0	
Sky Forest	PP	144	13.3	13.7	0.8	0.8	1.6	
University Conf. Center	PP	309	15.5	15.6	0.0	1.5	1.5	Moderate
Breezy Point	PP	236	16.3	16.0	2.7	2.7	5.5	
Camp Paivika	PP	217	17.1	16.4	0.0	3.1	3.1	
Dogwood A	PP	168	19.9	16.5[b]	1.2	0.0	1.2	
Tunnel Two Ridge	PP	122	19.5	16.7[b]	0.0	1.4	1.4	
Camp Angeles	PP	112	25.6	16.8[b]	0.0	1.5	1.5	
Barton Flats	PP	200	21.4	18.7[b]	3.7	3.7	7.4	
Barton Flats	JP	124	21.0	19.7[b]	3.6	3.6	7.3	
Snow Valley 2	JP	129	22.1	19.7[b]	0.0	1.0	1.0	
Green Valley Creek	JP	43	21.8	20.5[b]	0.0	1.5	1.5	
Camp Oceola	JP	192	21.7	22.8[b]	0.8	7.2	8.0	Slight
Bluff Lake	JP	186	29.4	31.8[b]	0.0	0.0	0.0	Very slight
N.E. Green Valley	JP	120	33.1	32.1	0.0	0.0	0.0	
Heart Bar	JP	130	44.0	39.2[b]	0.0	0.9	0.9	No visible symptoms
Sand Canyon	JP	56	41.3	47.3[b]	0.0	0.0	0.0	
Holcomb Valley	JP	193	46.4	47.7	0.0	0.0	0.0	

[a] Number of trees per hectare.

[b] Difference significant at probability of 0.05 (comparisons valid only between years at a single plot).

TABLE 12-4. CHANGES OF TIMBER VOLUME AND PERCENTAGE OF TOTAL JEFFREY PINES[a] AT BARTON FLATS IN THE SAN BERNARDINO NATIONAL FOREST[38]

Risk classes	1952 Timber volume, bd ft	1952 Percentage of trees	1963 Timber volume, bd ft	1963 Percentage of trees	1972 Timber volume, bd ft	1972 Percentage of trees
	Control plot 1 (JCA Camp, Highway 38)					
Total, all classes	73,040	100	63,530	100	52,730	100
Risks 1 and 2	58,520	87	38,700	73	23,780	55
Risk 3	6,740	7	14,630	13	14,140	16
Risk 4	7,780	5	10,200	7	14,810	20
Snags and current stumps[b]	1	1	11	7	13	8
	Control plot 2 (Camp Oceola Road)					
Total, all classes	120,130	100	112,660	100	112,930	100
Risks 1 and 2	110,830	93	98,080	82	45,670	32
Risk 3	5,990	3	10,170	6	37,420	30
Risk 4	3,310	2	4,410	6	29,840	28
Snags and current stumps[b]	3	2	13	6	18	10

[a] In four insect risk classes at two control plots excluded from sanitation salvage logging between 1952 and 1972.[38]

[b] Accumulation during 10-year period. Data obtained from the Supervisor's Office, San Bernardino National Forest.

exposure to ambient air. Indirect effects result from alterations in numbers or distribution of the plant and animal population exposed to ambient air. For example, if air pollution eliminates or decreases the quantity of a susceptible plant species, the food chain of the consumers that feed on it may break down. The result could be a simpler and less stable ecosystem, with fewer numbers and species of plants and animals.

The clinical and pathological effects of oxidant air pollutants on domesticated vertebrates have been studied in the laboratory. No major references to studies of these effects on free-ranging native species seem to exist. The possible interactions of vertebrates, determined by extrapolation from laboratory studies, are shown in Figures 12-11 through 12-13 for the San Bernardino Mountain ecosystem.

When changes occur in one part of an ecosystem, the intimate nature of the interrelationships results in changes in many other parts. Any factor that causes change in one component of a system potentially affects all subsystems of that ecosystem. The most important indirect effects of oxidant air pollutants on vertebrates are those resulting in changes in the habitat. Foremost among these effects are those on the vegetation and the successional patterns of

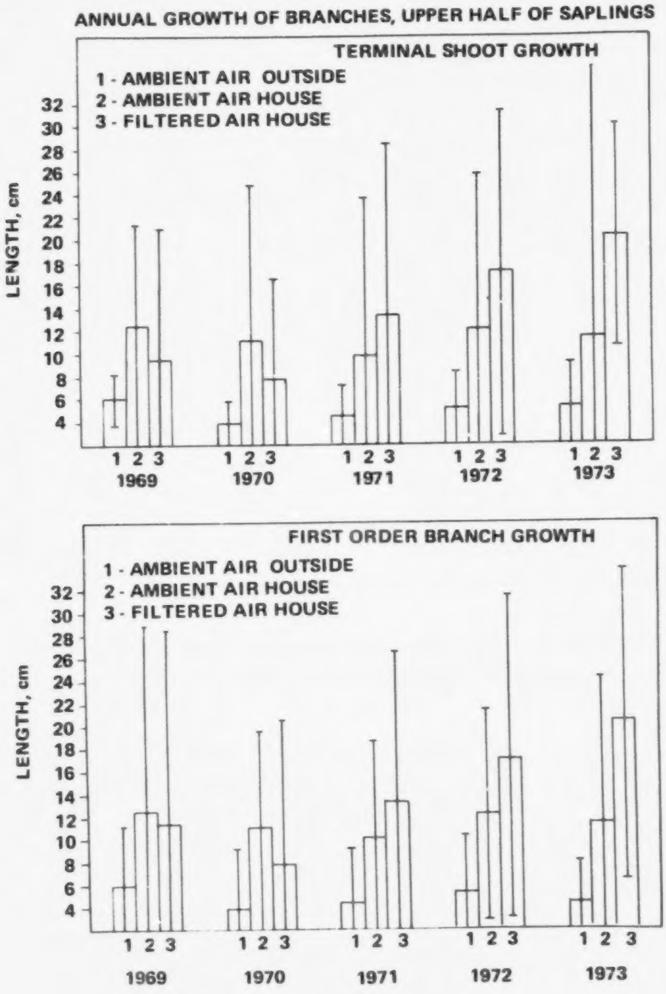


Figure 12-16. Annual growth of the terminal shoot (upper) and first-order branches (lower) in upper half of ponderosa pine saplings maintained in filtered or unfiltered (ambient) air greenhouses, or in outside ambient air (1968-73).[28]

the plant community. Because of the high degree of interrelationship and interaction of the vegetation, the fauna, and the inorganic matrix of an ecosystem, effects of air pollution on the vegetation potentially can result in changes throughout the ecosystem. Damage to vegetation is probably the most important effect of chronic, low-concentration air pollution on wildlife. Ponderosa pine, Jeffrey pine, and black oak are all susceptible to injury, and these are the most important trees within the forest as providers of food and habitat for wildlife. A similar selectivity by species doubtless occurs within the shrub and herb layers of the vegetation. The long-term effects will be (1) reduced production of fruits and seeds and (2) elimination of the sensitive plant species and a consequent reduction in the diversity of the vegetation. These effects will in turn lead to a reduction in the abundance and diversity of vertebrate fauna (in the numbers of pine-seed-eating squirrels, for example).

Likewise, Woodwell's prediction[71] of enhancement of the activity of insect pests and some disease agents (which has been demonstrated in this forest) could lead to an increase in vertebrate species that feed on invertebrates or utilize dead plants for cover. Birds would be the most likely to increase, as would (to a smaller extent) such small mammals as deer mice, which are partially insectivorous.

TABLE 12-5. TREE SPECIES AND SIZE COMPOSITION IN A STUDY AREA[a] AFFECTED BY OXIDANT AIR POLLUTION[38]

Tree size and class	Ponderosa pine	Incense cedar	White fir	Sugar pine
Understory, number/acre:				
Seedlings (up to 3.00 ft tall)	1057	2381	1043	302
Saplings (more than 3.01 ft tall, less than 3.99 in. dbh)	3	33	57	10
Poles (4.00 to 11.99 in. dbh)	21	12	38	3
As % of total	22.2	48.6	22.8	6.3
Overstory, number/acre:				
Standing (12.00 to 23.99 in. dbh)	18	9	8	3
Veteran (24 in. dbh and larger)	12	5	4	2
As % of total	49.6	22.7	19.7	8.0

[a] Trees within a 575-acre study area, San Bernardino National Forest, Calif.

The indirect effects of ozone through modification of the availability of food for insects, particularly in a conifer forest ecosystem, has received some investigation.[52,53] The weakening of ponderosa pines by chronic exposure to ozone makes them more vulnerable to successful infestation by pine bark beetles (*Dendroctonus brevicomis* and *D. ponderosae*). Figure 12-17 shows a positive relationship between degree of tree injury and frequency of bark beetle infestation and the relative frequency of attack by the two species of bark beetles.[52] The relationship (see Figures 12-10 through 12-13) between tree health and brood productivity and the population dynamics of bark beetles and their insect associates in infested trees is under investigation.[38,56] Pine bark beetles have been a constant threat to ponderosa pines in the San Bernardino Mountains for many years, since before the inception of oxidant air pollution injury.[54] Bark beetles are a key element in accelerating the modification of stand structure.

Plant Parasites and Symbionts

Ozone injury to needles of eastern white pine increased infection by *Lophodermium pinastri* and *Aureobasidium pullulans*.[14] These results suggest that leaf tissue of many species may become susceptible to fungi that are normally saprophytes but become parasites when circumstances permit.

Another way that oxidant air pollution could affect this ecosystem is through an alteration in forest moisture. Elimination of vegetation cover allows the exposed soil to dry more rapidly, which would affect soil-burrowing and soil-inhabiting vertebrates. Also, the lower moisture content could reduce or inhibit fruiting-body formation of fleshy fungi. These fleshy fungi are an important food source for tree squirrels and make up a third or more of their diet in some seasons. A reduction in this food source would doubtless result in an even greater utilization of conifer seeds and acorns, thus reducing further the reproductive capability of these trees and eventually limiting future food supplies for the squirrel population.

Beneficial mycorrhizal fungi infect the small feeder roots of trees and other plants. The resulting relationship is symbiotic and involves an intimate exchange of minerals and essential metabolites. The host tree benefits through increased efficiency of nutrient uptake from the soil. Any interruption or imbalance of the exchange of materials between the host root tissue and the fungus mantle surrounding it can have deleterious effects on the fungus and the host. Such stresses as air pollution injury to the host undoubtedly disrupt this balance.[25] The feeder rootlet system of ponderosa pines in the San Bernardino Mountains and those of eastern white pine have shown marked deterioration involving a decrease in numbers of mycorrhizal rootlets and their replacement by saprophytic fungi that decay the small rootlets.[43]

Root-infecting fungi such as *Armillaria mellea* and *Formes annosus* are generally more virulent pathogens when they encounter trees already weakened by other stresses. This observation has been made mostly in Europe, where sulfur dioxide was the principal pollutant.

EFFECTS ON DECOMPOSERS

Although some of the solar energy fixed by producer plants is released by the respiration of these plants and of animals, much of it is stored in dead organic matter until released by decomposer organisms at rates that vary greatly with place, season, and kind of organic matter. Generally, one-third or more of the energy and carbon fixed annually in the forests is contributed to the forest floor as litter (mostly leaves).[42] Because litter is generally related to the quantity of photosynthetic tissue in the ecosystem, it is a useful index of ecosystem productivity.

TABLE 12-6. INJURY THRESHOLDS FOR 2-HOUR EXPOSURES TO OZONE[64]

Species	Injury threshold (ppm ozone for 2 hr)
Grassland-oak community species:	
Trees and shrubs:	
Acer grandidentatum Nutt.	over .40
Acer negundo L.	over .25
Artemesia tridentata Nutt.	.40
Mahonia repens G. Don	over .40
Potentilla fruticosa L.	.30
Quercus gambelii Nutt.	.25
Toxicodendron radicans (L.) Kuntze	over .30
Perennial forbs:	
Achillea millefolium L.	over .30
Ambrosia psilostachya DC.	over .40
Calochortus nuttallii Torr.	over .40
Cirsium arvense (L.) Scop.	.40
Conium maculatum L.	over .25
Hedysarum boreale Nutt.	.15
Helianthus anuus L.	over .30
Medocagp satova L.	.25
Rumex crispus L.	.25
Urtica gracilis Ait.	.30
Vicia americana Muhl.	over .40
Grasses:	
Bromus brizaeformis Fish. & Mey.	.30
Bromus tectorum L.	.15
Poa pratensis L.	.25
Aspen and conifer community species:	
Trees and shrubs:	
Abies concolor (Gord. & Glend.) Lindl.	.25
Amelanchier alnifolia Nutt.	.20
Pachystima myrsinites (Pursh) Raf.	over .30
Populus tremuloides Michx.	.15
Ribes hudsonianum Richards.	.30
Rosa woodsii Lindl.	over .30
Sambucus melanocarpa A. Gray	over .25
Symphoricarpos vaccinioiders Rydb.	.30
Perennial forbs:	
Actaeu arguta Nutt.	.25
Agastache urticifolia (Benth.) Kuntze	.20
Allium acuminatum Hook	.25
Angelica pinnata S. Wats.	under .25
Aster engelmanni (Eat.) A. Gray	.15
Carex siccata Dewey	.30
Cichorium intybus L.	.25
Cirsium arvense (L.) Scop.	under .40
Epilobium angustifolium L.	.30
Epilobium watsoni Barbey	.30
Eriogonum heraclioides Nutt.	.30
Fragaria ovalis (Lehm.) Rydb.	.30
Gentiana amarella L.	over .15
Geranium fremontii Torr.	under .40
Geranium richardsonii Fisch. & Traut.	.15
Juncus sp.	over .25
Lathyrus lanzwertii Kell.	over .25
Lathyrus pauciflorus Fern.	.25
Mertensia arizonica Greene	.30
Mimulus guttatus DC.	over .25
Mimulus moschatus Dougl.	under .40
Mitella stenopetala Piper	over .30
Osmorhiza occidentalis Torr.	.25
Phacelia heterophylla Pursh	under .25
Polemonium foliosissimum A. Gray	.30
Rudbeckia occidentalis Nutt.	.30
Saxifraga arguta D. Don	under .30
Senecio serra Hook.	.15
Taraxacum officinale Wiggers	over .25
Thalictrum fendleri Engelm.	over .25
Veronica anagallis-aquatica L.	.25
Vicia americana Muhl.	over .25
Viola adunca Sm.	over .30
Annual forbs:	
Chenopodium fremontii Wats.	under .25
Callomia linearis Nutt.	under .25
Descurainia californica (Gray) O.E. Schuls	.25
Galium bifolium Wats.	over .30
Gayophytum racemosum T. & G.	.30
Polygonum douglasii Greene	over .25
Grasses:	
Agropyron caninum (L.) Beauv.	over .25
Bromus carinatus Hook. & Arn.	under .25

One of the predicted effects of pollutants on ecosystems suggested by Woodwell[71] is a reduction in the standing crop of organic matter, which would lead to a reduction in nutrient elements held within the living system. The evidence discussed earlier definitely shows that primary production is much lower in an ozone-stressed conifer forest ecosystem. This result would be anticipated in all natural ecosystems or agroecosystems under similar stress.

The reservoir of energy and mineral nutrients represented by litter is a very important resource in natural ecosystems with closed nutrient cycles. The growth of new green plant tissue depends on the slow release of nutrients by decomposer organisms. In agroecosystems geared for high production, litter is often removed or burned, and fertilizer is added to the soil; the nutrient cycle is open and subsidized.

In a conifer forest, litter production and decomposition release about 80 percent of the total minerals in the biomass of the stand; the remainder is retained in the living parts of the tree.[31] Standing dead material is not considered litter.

In terrestrial ecosystems, most decomposers occupy the mantle of litter on the surface layers of the soil, where they supply the necessary recycling mechanisms to convert dead plant or animal material into humus and eventually into minerals, gases, and water. Small animals, arthropods, fungi, and bacteria exist as a complex in intricate food chains in which they feed on dead material and on one another as well, ultimately releasing the mineral nutrients needed by the producer populations. Without the decomposers, some essential elements (such as calcium, phosphorus, and magnesium) would concentrate in the litter until the supply in the soil was depleted. Growth of green plants would then be seriously limited.

It is not known whether ozone, PAN, or other oxidants could have any direct influence on decomposer organisms other than fungi (see Chapter 11, section on responses of mosses, ferns, and microorganisms) in the litter layer. But there does appear to be a rapid flux of ozone to soil surfaces. The ozone flux to some kinds of surfaces constituting ecosystems (e.g., vegetation, soil, and water) has been determined by Aldaz,[1] who expressed the flux as molecules/cm^2-sec × 10. The relative fluxes into different surfaces, assuming an ozone concentration of 40 μg/m^3, were: Fresh water, 0.5; snow, 0.9; grass, 1.1; sand or dry grass, 5; and juniper bush, 10. Furthermore, it was found that bare soil destroyed about 75 percent more ozone when dry than when moist. The determination of ozone flux to surfaces may be a far more realistic measure of dose to living organisms than atmospheric concentration of ozone, according to Munn.[37]

In summary, it is anticipated that decreasing litter production by green plants experiencing pollutant stress would result in a similar reduction in the inventory of nutrient elements held within

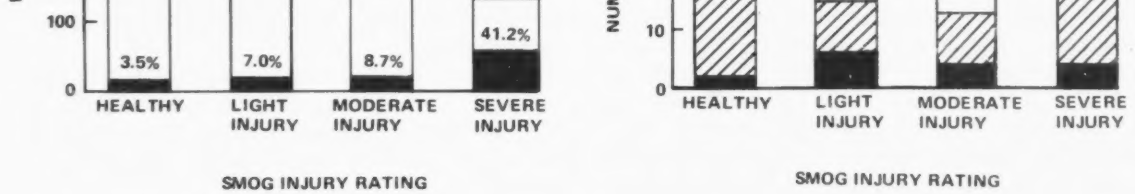


Figure 12-17. Relationship of degree of oxidant injury in ponderosa pines with bark beetle attack (left) and number of trees killed by western pine beetle, mountain pine beetle, or both species (right).[46]

the system, owing to the interruption of nutrient cycling pathways and mechanisms of nutrient conservation.[71,72]

Overall, it can be seen that subtle and simple initial changes may radiate and magnify throughout all trophic levels of the ecosystem. Restoration of the system may be impossible.

SUMMARY

Plants, animals, and microorganisms usually do not live alone but exist as populations. Populations live together and interact as communities. Communities, because of the interactions of their populations and of the individuals that constitute them, respond to pollutant stress differently from individuals. Man is an integral part of these communities, and as such, he is directly involved in the complex ecological interactions that occur within the communities and the ecosystem of which he is a part.

The stresses placed on the communities and the ecosystems in which they exist can be far-reaching, since the changes that occur may be irreversible. For example, it has been suggested that the arid lands of India are the result of defoliation and elimination of vegetation that induced local climatic changes not conducive to the reestablishment of the original vegetation.

An ecosystem (e.g., the planet Earth, a forest, a pond, an old field, or a fallen log) is a major ecological unit made up of living (biotic) and physical (abiotic) components through which the cycling of energy and nutrients occurs. A structured relationship exists among the various components. The biotic units are linked together by functional interdependence, and the abiotic units make up all of the physical factors and chemical substances that interact with the biotic units. The processes occurring within the biotic and abiotic units and the interactions among them can be influenced by the environment.

Ecosystems tend to change with time. Adaptation, adjustment, and evolution are constantly taking place as the biotic component, the populations, and the communities of living organisms interact with the abiotic component in the environment. Recognizable sequential changes occur. With time, populations and communities may replace one another. This sequential change, termed "succession," may result in climax communities. The latter, which are structurally complex and more or less stable, are held in a steady state through the operation of a particular combination of biotic and abiotic factors. The disturbance or destruction of a climax community or ecosystem results in a return to a simpler stage. Existing studies indicate that changes occurring within ecosystems in response to pollution or other disturbances follow definite patterns that are similar even in different ecosystems. The basic biotic responses to the disturbance of an ecosystem can thus be broadly predicted.

Diversity and structure are most changed by pollution as a result of the elimination of sensitive species of flora and fauna and the selective removal of the larger overstory plants in favor of plants of small stature. The result is a shift from the complex forest community to the less complex, hardy shrub and herb communities. The opening of the forest canopy changes the environmental stresses on the forest floor, causing differential survival and, consequently, changed gene frequencies in subcanopy species. Associated with the reduction in diversity and structure is a shortening of food chains, a reduction in the total nutrient inventory, and a return to a simpler and less stable successional stage.

It should be emphasized that ecosystems are usually subjected to a number of stresses at the same time, not just to a single perturbation (e.g., oxidant).

The effects of oxidants on the San Bernardino Forest graphically demonstrate the changes that occur in natural ecosystems, as discussed earlier. The San Bernardino Forest has been undergoing oxidant stress as a result of long-range transport from Los Angeles, 224 km (140 miles) away, since the early 1940's. Losses of ponderosa and Jeffrey pines, the overstory vegetation, have increased dramatically as pollutant levels have risen. Black oak has also suffered oxidant injury. An alteration in the composition of both plant and animal populations has resulted because of the death of the ponderosa and Jeffrey pines.

The interaction of pollutant and inversion layers at the heated mountain slope results in the vertical venting of oxidants over the mountain crest by upslope flow.

Oxidant concentrations ranging from 100 to 200 $\mu g/m^3$ (0.05 to 0.10 ppm) at altitudes as high as 2432 m (8000 ft), approximately 1033 m (3400 ft) above the mountain crest, were measured by aircraft.

The total oxidant concentrations have been measured continuously from May through

September since 1968 at the Rim Forest/Sky Forest monitoring station. During each of the first 7 years of monitoring, between June and September, the total number of hours with concentrations of 160 μg/m^3 (0.08 ppm) or more of ozone was never less than 1300 hours. The number of hours wherein the total oxidant concentration was 390 μg/m^3 (0.20 ppm) or higher increased from fewer than 100 in 1969 to nearly 400 in 1974. It is not uncommon to observe momentary oxidant peaks as high as 1180 μg/m^3 (0.60 ppm). The duration of oxidant concentrations exceeding 200 μg/m^3 (0.10 ppm) was 9, 13, 9, and 8. hr/day going from the lower to the higher altitude stations.

The most recent data firmly indicate that oxidant concentrations will either increase annually or oscillate around the mean of present high concentrations in the foreseeable future.

The transport of the urban plume from the coast northeastward to the mountains can be readily demonstrated. Because of this transport, the permanent vegetation constituting natural ecosystems receives a large chronic exposure, and the short-lived, higher-value vegetation constituting the agroecosystem of the Los Angeles coastal plain is subjected to injurious doses in intermittent, short-term fumigations. Each situtation has measurable economic and aesthetic effects, but on different time scales. The single-species agricultural ecologic system (the agroecosystem) has little resilience to pollutant stress; losses are sometimes immediate and occasionally catastrophic. The complex natural ecosystem is initially more resistant to pollutant stress, but the longer chronic exposures cause disruption of both structure and function in the system that may be irreversible.

Oxidant injury to the mixed conifer stands of the San Bernardino Mountains beginning in the early 1940's, as indicated above, is well advanced. A similar problem is developing in the forests of the southern Sierra Nevada. Both areas show direct and indirect effects on all subsystems of the forest ecosystem—producers, consumers, and decomposers.

In summary:

1. Ozone injury has limited the growth and caused the death of ponderosa and Jeffrey pines. An estimated 1,298,000 trees have been affected. Decrease in cone production has resulted in a decrease in reproduction. Black oak has also suffered injury from ozone.
2. Reduction in fruits and seeds that make up the diet of most of the common small mammals influences the populations of these organisms.
3. Essential processes, such as recycling of nutrients, may be disrupted, causing a limitation in the growth of vegetation.
4. Death of the predominant vegetation has caused an alteration in the species composition and a change in the wildlife habitat.

The San Bernardino Mountain study illustrates the complexity of the problems caused by environmental pollution. The changes that have occurred in this mountain ecosystem as a result of oxidant transport have already influenced the importance and value of this natural resource to the residents of southern California.[67]

The injury to the eastern white pine in the Appalachian Mountains resulting from oxidant transport from the urban northeast has begun a similar sequential change that could degrade this important recreational area. Total oxidant peaks as high as 220 μg/m^3 (0.11 ppm) were recorded for July 1975. Concentrations exceeding 160 μg/m^3 (0.08 ppm) were measured in June 1976. These episodes resulted in significant increases in oxidant injury to three categories of eastern white pine in the Blue Ridge Mountains.

Evaluating the contribution of functioning natural ecosystems to human welfare is a complex task and usually involves weighing both economic and social values. However, because natural ecosystems are life support systems, their value should not be quantified in economic terms.

With the passage of time, man has destroyed many of the naturally occurring ecosystems of which he was a part and has replaced them with simplified ecosystems wholly dependent on his care and protection and requiring a large input of energy.[50]

Man favors the simple, unstable and synthetic ecosystems, because when they are extensively managed and subsidized by the use of fossil fuels, they are highly productive. An agricultural ecosystem (agroecosystem) is an example of such a simplified ecosystem. The effects of oxidants on agroecosystems have been under study for more than 20 years. The study of effects on natural ecosystems is much more recent.

Plants grown in agroecosystems are largely annuals and can be replaced when they are susceptible to pollutant stress. Natural ecosystems remain in place year after year. Manmade pollutants are undoing relationships developed within these ecosystems over millions of years.

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13. EFFECTS OF OZONE ON MATERIALS

INTRODUCTION

Ozone is a major causative factor in the overall deterioration of many different types of organic materials. In fact, certain polymeric materials are more sensitive to ozone attack than are humans or animals. The magnitude of ozone damage, however, is difficult to assess because it is only one of the many photochemical oxidants in the atmosphere that contribute to the weathering of materials. Nevertheless, researchers have shown that ozone does accelerate the deterioriation of several classes of material, including elastomers (rubber), textile dyes and fibers, and certain types of paints. Also, a National Academy of Sciences report[28] goes into detail to show that the damage observed in most laboratory controlled experiments is caused by ozone rather than some other unmeasured oxidizing species.

That same document postulates that in urban atmospheres, other oxidizing species may be more damaging than ozone. It was assumed that the relative reaction rates of various oxidizing species with *trans*-2-butene (a gas) are the same for reactions with solid surfaces. This assumption did not consider that at ambient levels, reactions with surfaces will most likely be diffusion-controlled rather than activation-energy-controlled. This type of rate-controlling mechanism depends on the concentration gradient at the surface, which is roughly proportional to the ambient concentration. Ozone is the most abundant of the active oxidants.

MECHANISMS OF OZONE ATTACK

Ozone is so chemically active that, when concentrated, handling it becomes a problem because of its effect on ordinary materials. In general, materials of organic origin are affected deleteriously by concentrated ozone.[9] Bailey[1] has thoroughly reviewed the literature from 1939 to 1957 for reactions of ozone with a host of organic compounds, and he describes the reaction mechanisms in detail. Although it is incorrect to assume that all of these reactions would occur at atmospheric concentrations of ozone, it is possible that many of these reaction mechanisms would be operant.

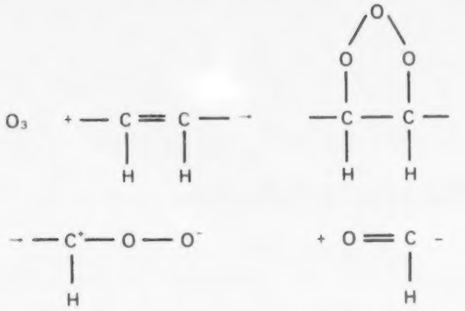
Many polymers are sensitive to atmospheric concentrations of ozone,[22] resulting in the occurrence of chain-scissioning or cross-linking or both. These reactions are related to the prevalence of double bonds in the polymer structure. Chain-scissioning results in a reduction in average molecular weight and in decreased tensile strength. Cross-linking increases the rigidity of some polymers, thus increasing brittleness and reducing elasticity.

Effects On Elastomers

OZONE CRACKING

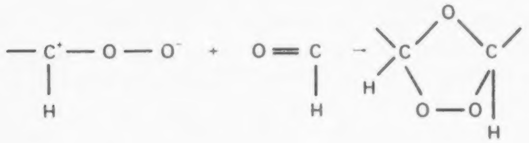
The cracking of stressed natural rubber exposed to outdoor environments has been a troublesome materials problem for many years. At first, most rubber technologists believed that sunlight was the major cause of this phenomenon. However, several investigators, mainly Williams[39] and Van Rossem et al.,[38] concluded that cracking was due to atmospheric ozone rather than sunlight, but the relative importance of ozone was not immediately recognized. Not until the 1940's did the research of Norton,[30] and especially that of Newton,[29] and Crabtree et al.,[10,11] suggest that free radicals, produced by the catalyzed photolysis of volatile peroxides present in smog, may also contribute to rubber cracking.

Cracking is related to the chemical structure of rubber compounds and their elastic properties. Vulnerable elastomers are those containing many double bonds. An example of a chemical mechanism may be written:[28]



(13-1)

The zwitterion and aldehydic group may combine to form a more brittle oxidized structure:



(13-2)

The most vulnerable compounds are natural rubber and synthetic polymers of styrene-butadiene, polybutadiene, and polyisoprene. These elastomers account for about 75 percent of the annual production value in the United States.

Except for natural rubber, the major use for these elastomers is in tires. Butyl, halogenated butyl, polychloroprene, vinyl-modified nitrile-butadiene, and carboxylated nitrile elastomers have some ozone resistance but required special formulation for optimum performance. Synthetic elastomers with saturated chemical structures such as silicones, ethylenepropylenes, chlorosulfated polyethylenes, polyacrylates, and fluorocarbons have inherent ozone resistance. These latter special-application materials, however, are relatively expensive and account for only a small fraction of the market on a weight basis.

Tensile stress is necessary to produce ozone cracking in sensitive elastomers. The resulting cracks develop at right angles to the direction of stress. Elastomers in a relaxed state can be exposed for long periods of time to relatively high concentrations of ozone without developing cracks. Crabtree and Malm[12] reported that cracks will develop in sensitive elastomers when strained as little as 2 to 3 percent and exposed to atmospheres containing only 20 $\mu g/m^3$ (0.01 ppm) ozone. As strain is increased to 100 percent plus, the number of cracks increases from a few deep cuts to numerous shallow ones. At still higher strain, small cracks are so numerous that the surface simply presents a frosted appearance, and damage from deep cracks is negligible.

Bradley and Haagen-Smit[7] summarized the factors that influence the action of ozone on elastomers. These include (1) the nature of the elastomeric compound, (2) degree of stress, (3) concentration of ozone, (4) time of exposure, (5) rate of the ozone contacting the elastomer, and (6) temperature. Furthermore, cracking may be divided into two main phases: crack formation and crack growth. Scientists have proposed several theories to explain ozone cracking, but the exact mechanism is still not clear. Braden and Gent[6] explained the strong dependence of crack growth on nominal tensile stress in terms of the Griffith theory of fracture mechanism. Fracture mechanics alone only partially explain observed relationships between number and size of cracks as a function of strain. Crack growth rate is also ozone-concentration dependent, which suggests that the rate of diffusion of ozone to the root of a crack is the rate-controlling factor.

More recently, Devries and Simonson[13] have applied the Griffith theory on a molecular scale. The energy required for crack growth is extracted from the elastic strain energy in the elastomer and is expressed in terms of the energy to rupture bonds and the number of bonds broken. They indirectly determined the cracking rates experimentally by measuring the number of resulting free radicals with a highly sensitive electron spin resonance (ESR) technique. Their observations were consistent with both theory and the results of previous damage studies.

ANTIOZONANTS

Fisher[17] and Mueller and Stickney[27] discussed the use of antiozonant additives to protect elastomers from ozone degradation. First developed in 1955, antiozonants are organic compounds, generally secondary aromatic amines and phenols. They are incorporated into elastomeric formulations during mixing and tend to migrate to the surfaces of parts after vulcanizing, where they form a film that chemically reacts with atmospheric ozone before it contacts the elastomer. These protective films are effective even when elastomers are stretched or flexed.

Although widely used in such elastomeric products as tires, conveyer belts, automotive parts, and cable insulation, antiozonants have limitations. They are expensive and add to the cost of finished products. For example, the addition of 1.5 percent antiozonants to automobile tires increases the cost by as much as $0.50 per tire. Furthermore, during product usage, antiozonants provide only temporary protection against ozone damage because they react with ozone and are inactivated. Contact with oils, gasoline, and other chemicals may also remove them by extraction.

The amount of antiozonant necessary for effective control increases with the anticipated concentration of atmospheric ozone. Although undocumented, several sources have told EPA scientists that tire manufacturers roughly double the amount of antiozonant added to rubber used in tires sold in California. Despite preventive measures, however, ozone cracking of sensitive elastomeric products still remains a major problem.

DOSE-RESPONSE DATA

In reviewing dose-response data, one must keep in mind the many factors that affect the rate of attack by ozone on vulnerable elastomers. Furthermore, methods of reporting effects data vary from one research to another and thus further complicate comparison. For example, an effect may be expressed as time required to initiate either microcracks or visible cracks, or it may be expressed as crackdepth or crackgrowth rate.

Much of the documented research on ozone-elastomer effects has been conducted at ozone concentrations considerably higher than those normally found in polluted environments. This has been intentional in order to accelerate reaction rates. However, some investigators have carried out research at lower ozone concentrations. Bradley and Haagen-Smit[7] evaluated a natural rubber formulation (Table 13-1) for susceptibility to ozone cracking. Strips were strained approximately 100 percent by bending. When exposed in a 13-mm-diameter glass tube to 40,000 mg/m^3 (2 percent or 20,000 ppm) ozone in air at a flow rate of 1.5 l/min, these specimens cracked instantaneously and broke completely within 1 sec; however, when they were exposed to various lower concentrations of ozone, different time periods were required for cracks to develop (Table 13-2). (Ozone levels were measured by the neutral KI method.) Based on these data, the initiation of cracks is controlled by the dose of ozone (concentration × time). The mean and estimated standard deviation of the dose for this particular experiment is a product of 1.32 ± 0.03 ppm × minutes.

TABLE 13-1. FORMULATION OF HIGHLY OZONE—SENSITIVE NATURAL RUBBER[a]

Ingredients	Parts by weight	% by weight
Rubber	100	35.91
Tire reclaim	125	44.88
SRF Black	33	11.85
Steric acid	1.5	0.54
Pine tar	8.4	3.02
Zinc oxide	4.7	1.69
Mercaptobenzothiazole	0.8	0.29
Diphenylguanidine	0.1	0.03
Sulfur	5	1.79

[a] Cured 40 min at 310,050 N/m^2 (45 psi) steam pressure.

TABLE 13-2. EFFECT OF OZONE ON NATURAL RUBBER[a]

Ozone concentration		Time to first sign of crack at 4× magnification, min
μg/m^3	ppm	
40	0.02	65
500	0.25	5
900	0.45	3

[a] See Table 13-1 for formulation; strained 100 percent.

Meyer and Sommer[25] exposed thin polybutadiene specimens, under constant load, to room air for which the average concentrations of ozone had been measured by the neutral KI method. Specimens exposed in the summer months to average ozone concentrations of about 96 μg/m^3 (0.048 ppm) failed by breaking into two separate parts after 150 to 250 hr. In the fall, at average ozone concentrations of 84 μg/m^3 (0.042 ppm), specimens failed between 400 and 500 hr. In the winter, at average ozone concentrations of 48 μg/m^3 (0.024 ppm), failures occurred between 500 and 700 hr. These data show the strong dependence of cracking rate on average concentrations of ozone. It takes much longer for complete failures to occur than it does to initiate cracks. The mean and estimated standard deviation of the dose required to cause 100 percent complete failures in these specimens were 996 ± 270 ppm × minutes.

Edwards and Storey[16] determined the effects of ozone on elastomeric formulations (Table 13-3) containing various amounts of antiozonants and either a styrene-butadiene rubber (SBR) polymer that had been polymerized at elevated temperatures ("hot" SBR) or one polymerized at room temperatures ("cold" SBR). The basic polymers were used in tire sidewall formulations. The investigators exposed test specimens under strain (100 percent) to ozone concentrations (measured by the neutral KI method) of 490 ± 100 μg/m^3

(0.25 ± 0.05 ppm) at 49°C (120°F). Table 13-4 shows the calculated rate of cracking for the various formulations. The results provided evidence that the hot SBR polymer is inherently superior in resistance to attack by ozone, and increasing amounts of antiozonants produced significant reductions in the rate of cracking and thus increased the dose necessary to initiate visible cracks. The function is exponential:

$$\text{Dose (ppm} \times \text{minutes)} = a\,e^{bz} \qquad (13\text{-}3)$$

where z is percent antiozonant. Regressions on these data show that b is essentially the same for the two formulations (1.5) and that the a values are 17.3 and 9.8, respectively, for the hot and cold SBR. The dose would have to be three orders of magnitude greater for cracks to grow to 2.54 mm. To prevent failure of 5-mm-thick sidewalls exposed under 100 percent strain at 49°C (120°F) to 100 $\mu g/m^3$ ozone for 3 years (781,840 ppm × minutes), an estimated 0.56 percent and 0.94 percent of antiozonant in the hot and cold SBR would be needed, respectively.

TABLE 13-3. TIRE SIDEWALL FORMULATION[16]

Ingredients	Parts by weight
Polymer (hot or cold SBR[a])	100
Circosol 2 XH	10
FEF black	30
SRF black	10
Zinc oxide	3
Steric acid	2
Antiozonant (Santoflex AW)	Variable
Crystex	2

[a] SBR is styrene-butadiene rubber polymerized either at high or room temperature.

TABLE 13-4. EFFECTS OF OZONE ON SIDEWALL FORMULATIONS CONTAINING VARIOUS ANTIOZONANT CONCENTRATIONS

Polymer	Antiozonant concentration, %	Rate of cracking		Time to first sign of crack, min[a]
		$\mu m/hr$	in./hr × 10^{-4}	
Hot SBR	0	2.34	0.92	65
	0.32	1.75	0.69	87
	0.63	0.89	0.35	170
	1.25	0.33	0.13	460
Cold SBR	0	4.01	1.58	38
	0.32	2.16	0.85	71
	0.63	1.45	0.57	105
	1.25	0.61	0.24	250

[a] Added to enable comparison with data in Table 13-2. First sign of crack was assumed to be 2.54 μm (1×10^{-4} in.) crack depth, visible at 4× magnification. Data are not found in the paper by Edwards et al.[16]

Hofmann and Miller[21] found that the behavior of rubber exposed to ozone under laboratory conditions correlated well with the service behavior of tires in localities where atmospheric ozone concentrations were high. The relative susceptibility of different formulations of white sidewall rubber remained the same, whether exposed under laboratory conditions to as much as 1000 $\mu g/m^3$ (0.5 ppm) ozone, or in the ambient air of the Los Angeles areas. The rate of cracking is thus a function of ozone concentration.

In a factorial-design, controlled-environment experiment, Haynie et al.[19] exposed several classes of materials to realistic levels of pollutants and climatic factors. White sidewall specimens from a top-quality steel-belted radial tire were exposed (strained at 10 and 20 percent) for up to 1000 hr. The level of ozone was a statistically significant factor in the rate of cracking of the white sidewall rubber. The average results with respect to strain and ozone level are given in Table 13-5.

TABLE 13-5. CRACKING RATES OF WHITE SIDEWALL TIRE SPECIMENS[19]

Ozone conc., $\mu g/m^3$ (ppm)	Strain, %	Mean cracking rate ± S.D.,[a] mm/yr
160 (0.08)	10	10.36 % 7.76
	20	11.70 % 7.22
1000 (0.5)	10	19.80 % 9.64
	20	24.09 % 6.24

[a] S.D. = estimated standard deviation of the mean.

Cracking rates are not directly proportional to ozone concentrations for these two levels. A coefficient for estimating crack depth based on the lower concentration is 34.5 ± 4.8 mm/ppm-year. This indicates it would take 3 years to crack a 5-mm sidewall exposed to 100 $\mu g/m^3$ (0.05 ppm). This does not include the time required for the resulting exposed tire cord to fail.

For this particular premium tire, one would expect the tread to wear out before sidewall failure from ozone damage occurred. However, the casing would have questionable value for retreading.

ECONOMICS

As the tread wear on passenger car tires improves, more or better antiozonants will have to be added to sidewall rubber formulations to prevent cracking from becoming the limiting factor in tire life. Thus part of the cost of premium tires will be the result of increased protection against atmospheric ozone.

Mueller and Stickney,[27] on the basis of a rubber industry survey, assessed the economic impact of air pollution on elastomeric products in the United States. They estimated the loss at the consumer level to be $500 million annually. About $170 million of this total was associated with various means of protection, including antiozonants and special polymers. The remaining $330 million was associated with premature failure of rubber products. (See Reference 28 for a more complete description of technique and costs.) The respective annual per capita costs in 1970 are $0.85 and $1.65. If more were spent on preventive measures, there would be less cost for failures. The relationship between dose to initiate cracks and antiozonant level suggests that for a fixed dose in 1970, failure costs (Y) would be related to preventive costs (X) by the relationship

$$Y = 5.9\ e^{-1.5X} \qquad (13\text{-}4)$$

The total cost is the sum of X and Y. By differentiating with respect to (X) and setting it equal to zero, the optimum total cost in 1970 is calculated to be $2.12. This value is $0.38 less per person than was estimated.

By reducing ambient levels of ozone, it is possible to establish some new optimization of preventive and failure costs. Assuming that a population-weighted national annual average for ozone was 65 $\mu g/m^3$ in 1970, the optimum per capita cost as a function of average ozone concentration in the ambient is:

$$\text{Cost} = 0.66\ (\ln O_3\ \ 1) \qquad (13\text{-}5)$$

when O_3 concentration is $\mu g/m^3$ annual average.

Because it is unlikely that consumers will optimize present prices with anticipated use life, these costs serve as a minimum estimate. A cost function with a 25-percent error range is

$$\text{Cost} = \$0.88\ (1 \pm 0.25) \times (\ln O_3\ \ 1) \quad (13\text{-}6)$$

This relationship suggests that if the national annual average concentration of ozone were lowered from 65 $\mu g/m^3$ to 50 $\mu g/m^3$ there could be a savings in elastomer damage and damage prevention costs of from 37 to 61 million dollars. Comparably, lowering the annual average from 100 $\mu g/m^3$ to 50 $\mu g/m^3$ for 6.9 million people in the Los Angeles Standard Metropolitan Statistical Area (SMSA) could save from 2.0 to 3.3 million dollars.

Effects on Textiles

Both fibers and dyes can be damaged by ozone; however the fading of dyes is a more significant factor than the loss of fiber strength.

DYE FADING

Although atmospheric nitrogen oxides were identified during the early 1900's as an important cause of color fading in certain textile dyes, mainly disperse dyes on acetate fabrics, it was not until the mid-1950's that the research of Salvin and Walker[32] showed the effects of atmospheric ozone. This discovery came about as a result of field testing acetate fabrics containing newly synthesized blue disperse dyes. Previous laboratory exposures of these dyes had shown them to be highly resistant to fading by nitrogen oxides. Field testing was carried out to determine if the performance of these new dyes was as good in actual use as in laboratory tests.

The researchers exposed fabrics dyed with one of the new blue dyes (as a color component) in homes located in areas known to have either low or relatively high levels of nitrogen oxides. At the low-nitrogen-oxides sites, they found that fading for many of the fabrics equaled, and in some cases exceeded, the fading observed at the higher-nitrogen-oxides site. Much of the observed fading was characterized by a bleached, washed-out appearance rather than the familiar reddening that nitrogen oxides produce in most sensitive dyes. This anomalous behavior suggested that some other oxidizing agent was responsible and was present at all exposure locations.

To describe this fading phenomenon, the investigators coined a new term, "O-fading." Follow-up laboratory investigations and general observations showed atmospheric ozone to be responsible. Ozone concentrations of 200 $\mu g/m^3$ (0.10 ppm) produced marked fading in most of the blue disperse dyes and in some of the reds and yellows.

The discovery of the O-fading was useful in explaining much of the anomalous fading of certain dyed fabrics observed during subsequent lightfastness testing and service exposure trials reported by Schmitt.[34,35] Other types of dyes besides the disperse dyes showed abnormal fading. Furthermore, the results of the service trials emphasized the importance of relative humidity, suggesting that the increased moisture content of fibers such as cotton promoted and accelerated the absorption and reaction of pollutants with vulnerable dyes. They also alerted the textile industry to the possibility of potential consumer complaints. Such complaints began to appear in the early 1960's and concerned mainly two distinctly different textile materials—polyester-cotton/permanent press fabrics, and nylon carpet.

Salvin[31] reported that the fading of polyester-cotton/permanent press fabrics was first noted on the folds and edges of slacks stored in warehouses or on the stock shelves of retail outlets. Some garments faded after storage in warehouses for as few as 10 days. Incidents occurred in various locations including California, Texas, and Tennessee. Because of the volume of garments involved, some producers suffered heavy economic losses. Fading was marked by a loss in blue along with a slight increase in red, suggesting that ozone and, to a lesser degree, nitrogen oxides may have been the active fading agents, since sunlight was obviously not a factor. This conclusion did not seem reasonable, however, because the cotton was dyed with vat dyes and the polyester with disperse dyes, and both dye-fiber systems are generally resistant to fading by common air pollutants. Furthermore, fading occurred only on fabrics that had been made into finished products and then cured to set the permanent press resin. Fading had not been observed during the temporary storage of dyed fabrics either before or after treatment with permanent press resins (before curing).

However, after a thorough investigation, including extensive laboratory tests, researchers found ozone to be the major fading agent, with nitrogen oxides also capable of causing fading, but to a lesser extent. The fading mechanism, which is unique and complex, takes place as a result of the curing operation and involves the disperse dyes on the polyester fibers rather than the vat dyes on the cotton. During curing, some disperse dyes partially migrate to the permanent press finish, which is a combination of reactant resin, catalysts, softeners, and nonionic wetting agents. The disperse dyes migrate to the solubilizing agents (nonionic surfactants and softeners) in the finish and are then in a medium in which fading by air contaminants can easily occur. Softeners are an especially good medium for absorbing gases.

The choice of catalyst used in the finish plays an important role, as the migration of disperse dyes increases significantly when magnesium chloride is used as a catalyst rather than zinc nitrate. Magnesium chloride is capable of forming complexes with certain anthraquinone disperse dyes (blues and reds), and these complexes are soluble in the resin finish. Therefore, as Dorset[15] pointed out, to eliminate this fading problem, textile processors must carefully select materials that make up the permanent press finish and/or replace vulnerable dyes with those that resist migration.

According to Salvin,[31] the fading of nylon carpets originated mainly in the warm humid areas from Texas to Florida and, as a result, became known as "Gulf Coast fading." In one case, nylon carpeting in a Texas apartment complex showed visible fading only 30 days after installation. A few similar incidents were also noted along the east coast and in the Los Angeles area. Fading occurred on carpets manufactured from both nylon 77 and nylon 6 fibers. Disperse dyes were used because many possess easy leveling properties necessary to avoid dye streaks. Fading took place largely on those carpets dyed with Disperse Blue 3 as one of the color components. Avocado, a tertiary-dyed, dull green shade, was a particularly sensitive color. The fading of this color was characterized primarily by loss in blue, which caused the green color to turn gradually to a dull orange shade.

Investigators eliminated sunlight as a primary cause of fading, since the phenomenon occurred in rooms where light intensity was low. Exposure of carpet samples to standard test methods for ozone also failed to duplicate the color change. Since fading complaints occurred in humid environments, and since previous lightfastness testing and service trials had established that conditions of high relative humidity promoted fading of certain dyes by ozone, the investigators next exposed carpet samples to ozone in the presence of high relative humidity (85 to 90 percent). Under these conditions, pronounced fading took place on those samples containing Disperse Blue 3, and the fading was similar to the color changes observed in homes along the Gulf Coast. Later experiments established that the relative humidity must be somewhat above 65 percent for pronounced ozone fading to occur. The fading problem may be prevented by using dyes that are more ozone resistant and by using nylon fibers that have been modified (by dry-heat texturing) to decrease the accessibility and diffusion rate of ozone.

Complaints were also received from consumers about faded drapery and upholstery fabrics. Salvin[31] reported that the combination of ozone and high humidity causes pronounced fading of certain dyes on cotton and rayon fabrics.

Beloin[3,4] investigated the effects of air pollution on various dyed textiles by conducting both field and controlled-environment laboratory studies. The field study consisted of exposing a wide range of dyed fabric in light-tight cabinets located at four urban and four rural (control) sites that were nearby and had similar characteristics except for air pollution. These sites represented a cross section of various types of pollution (urban sites) and climates. The study was carried out over a 2-year period, with eight consecutive 3-month seasonal exposures.

Color change data, along with air pollution and weather measurements, were statistically analyzed in an effort to identify the factors that caused fading. Beloin found that about two-thirds of the fabrics studied showed appreciable fading. Most of these fabrics faded significantly more at urban sites than at the rural sites, and the amount of fading varied among metropolitan areas and among seasons. Air pollution was assumed to account for most of the environmental differences between the urban and the corresponding rural control sites. Analysis suggested that the pollutants measured (NO_2, ozone, and SO_2) appeared to be those most responsible for fading. Generally it was impossible to separate the effects of individual pollutants because they were confounded with the effects of other pollutants.

The controlled-environment laboratory study was designed to assess the effects of common air pollutants, temperature, and relative humidity on the colorfastness of 30 dyed fabrics selected from those exposed during the field study. Fabric samples were exposed to two concentrations of ozone: 100 $\mu g/m^3$ (0.05 ppm) and 1000 $\mu g/m^3$ (0.50 ppm). Ozone concentrations were continuously measured by coulometric (Mast) analyzers. As might be expected, under similar exposure conditions, high ozone levels produced significant fading in more fabric samples than did low levels. Low levels, nevertheless, produced visible fading in about one-third of the sensitive fabrics—an important finding since the low levels were similar to those frequently found in metropolitan areas. The study also demonstrated that high relative humidity (90 percent) and, to a lesser extent, high temperature (32°C or 90°F) are significant factors in promoting and accelerating ozone-induced fading, thus confirming what investigators observed during previous service trials. The amount of ozone fading as a function of time was dependent on such factors as color and type of dye, fiber substrate, and environmental conditions.

Haynie et al.[19] and Upham et al.[37] exposed three moderate-to-high-volume-usage drapery fabrics to combinations of controlled-environment conditions to determine both direct and synergistic effects of gaseous pollutants. There were no statistically significant direct or synergistic effects of ozone on the fading of any of the dyes after exposures of 1000 hr to ozone up to 1000 $\mu g/m^3$. Relative humidity was a significant fading factor for all three fabrics, and nitrogen dioxide faded one of the fabrics. Fading as a function of time was consistent with the theoretical relationship:

$$\Delta E = \Delta E_m(1 - e^{-at}) \qquad (13\text{-}7)$$

where ΔE_m represents the maximum fade or complete dye destruction, t is time, and a is a constant containing the effects of environmental variables.

An unacceptable level of fading is subjective and varies considerably with individuals. A percentage of useful life lost depends only on the ozone level, because other factors, including subjective unacceptable levels of fading, cancel out. This relationship is:

$$\text{Percentage life lost} = \frac{(a_1 - a_0)}{a_1} \quad 100 \qquad (13\text{-}8)$$

where a_0 is the exponential coefficient for clean air, and a_1 is the value with some level of pollution. These functions should be applicable to ozone fading as well as NO_2 fading.

Haylock and Rush[18] studied ozone fading of anthraquinone dyes on nylon fibers in controlled environments. Olive I and Olive II dyes on nylon 6 carpets yarn was exposed to 400 $\mu g/m^3$ and 1800 $\mu g/m^3$ ozone at 40°C (104°F) and 90 percent relative humidity. The same materials were exposed to 400 $\mu g/m^3$ ozone at 40°C (104°F) and 70 percent and 80 percent relative humidity. C.I. Disperse Blue 3 and C.I. Disperse Blue 7 on nylon 6 were exposed to 400 $\mu g/m^3$, 1000 $\mu g/m^3$, and 2000 $\mu g/m^3$ ozone at 40°C (104°F) and 90 percent relative humidity. They were also exposed at 50°C (122°F) and 30 percent relative humidity to 400 $\mu g/m^3$ ozone.

The fading curves were highly consistent with the theoretical relationship of equation 13-7. Regression equations on the Haylock and Rush[18] data can account for 99 percent of the variability. With these data, coefficients were calculated for three of the dyes that were significantly affected by different ozone levels. Linear regressions of these coefficients with respect to ozone were then calculated. For the exposures at 40°C (104°F) and 90 percent relative humidity, the results are as follows:

Olive I:

$$a = 0.381 + 3.65 \times 10^{-5}\, O_3 \qquad (13\text{-}9)$$

Olive II:

$$a = 0.00107 + 1.18 \times 10^{-5}\, O_3 \qquad (13\text{-}10)$$

C.I. Disperse Blue 7:

$$a = 0.0212 + 5.35 \times 10^{-6}\, O_3 \qquad (13\text{-}11)$$

where a is reciprocal hours, and O_3 is in $\mu g/m^3$. Assuming that the relative effect of ozone with respect to other fading mechanisms remains the same at normal temperatures and relative humidities, these relationships convert to the following.

Olive I:

$$\text{Percent life lost} = \frac{0.096\, O_3}{1 + 0.00096\, O_3} \qquad (13\text{-}12)$$

Olive II:

$$\text{Percent life lost} = \frac{1.1\, O_3}{1 + 0.011\, O_3} \qquad (13\text{-}13)$$

C.I. Disperse Blue 7:

$$\text{Percent life lost} = \frac{0.025\, O_3}{1 + 0.00025\, O_3} \qquad (13\text{-}14)$$

These functions curve only slightly when ozone is below 100 $\mu g/m^3$. Thus the costs of early replacements can be assumed to be directly proportional to ozone levels.

FIBERS

Because cellulose fibers are vulnerable to oxidation, atmospheric ozone is a potential cause of degradation. With this in mind, Bogaty et al.[5] carried out experiments to study the possible role of ozone in the deterioration of cotton textiles. They exposed samples of duck and print cloth to air containing between 40 and 120 $\mu g/m^3$ (0.02 and 0.06 ppm) ozone (measured by the neutral KI method at room temperature and in the absence of light). Samples were exposed both dry and wet; the moisture content of the wet samples was never less than 50 percent. The researchers used changes in fluidity values to assess degradation. Fluidity (reciprocal of viscosity) is an indirect measure of the average molecular weight of polymer. Such reductions reflect changes in tensile strength and other physical properties.

After the cloth samples were exposed for 50 days, fluidity values increased for the cotton samples that were moist during exposure, but they did not appreciably change for the samples that remained dry during exposure. Fluidity values for control samples that were kept moist but not exposed to ozone showed little change. These results indicate that ozone caused virtually all the degradation. In addition to increased fluidity, the moist samples experienced a 20-percent loss in tensile strength after exposure to ozone. Similar fabrics were also exposed to higher levels of ozone, resulting in a greater increase in the degradation (fluidity) of the moist samples. The overall study showed that low levels will degrade cotton fabrics if they are sufficiently moist. The mechanism of ozone attack appears to be primarily due to its increased solubility in water, since at room temperature, ozone is considerably more soluble in water than is air.

The investigators estimated that the laboratory exposure conditions (50 days, high moisture and ozone) were equivalent to 500 days of exposure under field conditions. They concluded that ambient levels of ozone, while capable of damaging moist cotton fabrics, produce deterioration that was slight compared with that from other elements of weathering such as sunlight, heat, alternate wetting and drying, and micro-organisms.

Morris[26] also studied the effects of ozone on cotton. Samples of a special print cloth were exposed in the absence of light to 1000 $\mu g/m^3$ (0.5 ppm) ozone (coulometric method, Mast analyzer) for 50 days in a chamber maintained at 21°C (70°F) and 72 percent relative humidity. No appreciable effect on breaking strength or fluidity was found. Apparently, the moisture content of the cotton (9 percent) was not high enough to produce the degradation that Bogaty measured in wet cotton samples, even though the concentration of ozone was considerably higher (about 10 times).

The laboratory study of Kerr et al.[23] examined the effect of periodic washing on the tendering of cotton fabrics exposed to ozone. They exposed samples of print cloth, dyed with C.I. Vat Blue 29, in a chamber to a continuous supply of clean air with known levels of ozone added. The concentration of ozone (coulometric method, Mast analyzer) averaged 1500 $\mu g/m^3$ (0.75 ppm), and samples were exposed at room temperature in the absence of light. The investigators did not measure relative humidity, although they attempted to increase the humidity by placing a pan of water on the chamber floor. At 3-day intervals, the cotton samples were removed from the chamber. Half of them were machine-washed, and the other half were soaked in water for 1 min. All samples were passed through a hand wringer to remove excess water before they were returned to the chamber for further exposure. Control samples were kept in a light-tight box maintained at 21°C (70°F) and 65 percent relative humidity, and they were given the same washing and soaking treatment.

After an exposure period of 60 days, which included 20 washing and soaking treatments, the change in strength of the control fabrics was not significant. By comparison, the fabrics exposed to ozone changed significantly; the loss in strength for the washed fabrics was 18 percent, and for the soaked fabrics, 9 percent. The investigators attributed these losses to ozone.

Since Morris[26] found no degradation under exposure conditions similar to those used by Kerr, the washing and soaking treatment would appear to affect in some way the sensitivity of the fabrics to ozone. Nevertheless, when one attempts to equate Kerr's findings with actual conditions encountered by consumers, the degradation seems minimal in view of the fact that average levels of ozone under field conditions are less than 10 percent of the levels used in the laboratory exposure.

In laboratory studies, Zeronian et al.[40] simultaneously exposed modacrylic (Dynel), acrylic (Orlon), nylon 66, and polyester (Dacron) fabrics to artificial sunlight (xenon arc) and charcoal-filtered clean air contaminated with 400 $\mu g/m^3$ (0.2 ppm) ozone (analytical method not given) at 48°C (118°F) and 39 percent relative humidity. During exposure, the fabric samples were sprayed with water for 18 min every 2 hr. Ozone damage was measured by comparing these samples with fabrics exposed to the same environmental conditions but without ozone. After exposure for 7 days, the investigators found that ozone did not affect the modacrylic and polyester fibers; but it did seem to affect the acrylic and nylon fibers slightly.

ECONOMICS

Barrett and Waddell[2] used data from an unpublished contract report by Salvin to estimate the national cost of ozone fading at approximately $80 million/year. These costs were associated with the fading of acetates/triacetates, nylon carpets, and permanent press fabrics. Salvin produced his estimates from costs of preventive measures as well as loss-of-use life based on an industry survey. Preventive measures included more expensive dyes and manufacturing procedures, research, and quality control testing. The estimated totals for preventive and failure costs were $43 and $37 million, respectively. On a per capita basis, these respective annual costs are $0.22 and $0.18.

Because there was a lack of dose-response data, Barrett and Waddell[2] did not attempt to establish an economic damage function based on these estimates. One can be postulated assuming an analogy with elastomer economic damage costs. For elastomers, an estimate of the amount of damage saved by preventive measures is 5 times the cost of those measures. An analogous relationship between damage costs (Y) and preventive cost (X) is:

$$Y = 1.28e_{-8.9x} \qquad (13\text{-}15)$$

It follows, using the same procedures as with elastomers, that a reasonable economic damage function with 50 percent error is:

$$\text{Cost} = \$0.22\ (1 \pm 0.5)\ (1\text{n}\ O_3 - 0.74) \qquad (13\text{-}16)$$

over the range of ozone concentrations greater than 5.7 $\mu g/m^3$.

This relationship suggests that from $6.2 to $18.6 million could be saved annually by lowering the national annual average ozone concentration from 65 to 50 $\mu g/m^3$.

Effects on Paints

DAMAGE FUNCTION

Campbell et al.[8] have conducted laboratory research and field studies indicating that ozone may damage paint. The technique used to assess damage, erosion measurements (weight loss converted to thickness loss), proved to be the most meaningful. Erosion, or the gradual weathering of the surface of paint film, is the normal mechanism of failure that limits the service life of well formulated and properly applied exterior coatings.

The researchers selected five exterior coatings from four commercially important paint types: house paints (oil and acrylic latex), coil coating* finishes (urea-alkyd), automotive refinishes (nitrocellulose/acrylic), and industrial maintenance coatings (alkyd).

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*Coil coating is a factory-applied coating to a coil of sheet metal.

Panels coated with the different exterior paints were exposed to controlled-environment conditions, including clean air (control), clean air containing 200 μg/m^3 (0.1 ppm) ozone, and clean air containing 2000 μg/m^3 (1.0 ppm) ozone. Ozone concentrations were measured using the buffered KI method. The exposure chamber operated on a 2 hr light-dew cycle consisting of 1 hr of xenon light, 70 percent relative humidity, and a black panel temperature of 66°C (151°F), followed by 1 hr of darkness at 100 percent relative humidity of 49°C (120°F), during which time moisture condensed on the coated panels. One group of panels was exposed to simulated sunlight while a like group of panels was shaded during the exposures.

Erosion measurements were made after exposure periods of 0, 400, 700, and 1000 hours. Erosion rates for each paint type and exposure condition were then calculated from the erosion results. Zero-hour results, however, were excluded from these calculations because erosion rates during the 0- to 400-hr period were greater than during the remaining exposure (400 to 1000 hr). The researchers concluded that this was primarily caused by removal of water soluble materials and that materials subsequently eroded consisted mainly of binder and pigment.

Generally, exposures to 2000 μg/m^3 (1 ppm) ozone produced statistically significant increases in erosion rates compared to clean air (zero pollution) conditions. Erosion rate increases, however, varied considerably among paint types. Oil-based house paint experienced the largest erosion rate increase; industrial maintenance paint, a moderate increase; and latex and coil coatings and automotive refinishes the smallest increases. As would be expected, unshaded panels eroded more (degradation by sunlight) than shaded panels. Because of considerable data variability, exposures to 200 μg/m^3 (0.1 ppm) ozone generally did not produce statistically significant erosion rate increases over clean air exposures.

Table 13-6 gives regression coefficients for the effects of ozone based on the data of Campbell et al.[8] Because the rate of ozone damage is controlled by the rate of ozone transport to the paint rather than activation energy for reaction, these coefficients should be applicable to lower ambient temperatures.

All of the coefficients are positive, and half are statistically significant. The largest coefficients were determined on data with the greatest variance. Consequently, they are not statistically significant, although the effects they represent could be real.

Field exposures were conducted in Leeds, N.Dak., Valparaiso, Ind., Chicago, Ill., and Los Angeles, Calif. These locations, respectively, represent clean, moderate-SO_2, high-SO_2, and high-ozone environments.

Panels of the five different coatings were mounted at an angle of 85° from the horizontal to represent normal exposure conditions of house siding. Half of the panels were mounted facing south, and half facing north. Panels were evaluated after 0, 3, 7, and 14 months of exposure. As with the laboratory data, the zero-month results were not used in calculating erosion rates.

TABLE 13-6. PAINT EROSION RATE COEFFICIENTS FOR THE EFFECTS OF OZONE IN LABORATORY-CONTROLLED ENVIRONMENTS[36]

	Mean and estimated S.D. of erosion rate coefficients for ozone, μm/yr per μg/m^3	
Coating	Shaded	Unshaded
Automotive	0.00216 ± 0.00043[a]	0.00325 ± 0.00026[a]
Latex	0.00099 ± 0.00074	0.00214 ± 0.00007[a]
Industrial maintenance	0.01073 ± 0.01473	0.01098 ± 0.01774
Coil coating	0.00129 ± 0.00207	0.00752 ± 0.00176[a]
Oil house paint	0.00812 ± 0.00378[a]	0.02726 ± 0.01894

[a] Coefficient significantly greater than zero at 0.95 probability level.

The erosion rates for Leeds and Los Angeles are presented in Table 13-7. With the exception of the north-facing automotive coating, all of the erosion rates are significantly higher in Los Angeles than in Leeds. The latex, coil coating, and oil house paint contain fillers that are susceptible to SO_2 attack. The automotive and industrial maintenance coatings do not contain fillers. Thus, many of the differences in erosion rates between Leeds and Los Angeles for the filler-containing paints could be caused by differences in SO_2 levels. The observed differences in the automobile and industrial maintenance coatings could be attributed primarily to higher ozone levels in Los Angeles. For example, using the laboratory-obtained coefficients, ozone differences of 85 to 111 μg/m^3 can account for the erosion rate difference between Leeds and Los Angeles. Ozone levels were not measured, but an average difference of 40 to 60 μg/m^3 ozone is consistent with reported air pollution data. Also, average differences in temperature and other pollutants would be expected to contribute to the higher erosion rates in Los Angeles.

TABLE 13-7. PAINT EROSION RATES AT FIELD EXPOSURE SITES

Coating	Mean and estimated S.D. of erosion rates, μm/year			
	Panel facing north		Panel facing south	
	Leeds	Los Angeles	Leeds	Los Angeles
Automotive	0.305 ± 0.076	0.305 ± 0.046	0.305 ± 0.107	0.610 ± 0.031
Latex	0.305 ± 0.046	0.914 ± 0.137	0.305 ± 0.076	0.914 ± 0.351
Industrial maint.	0.914 ± 0.198	1.829 ± 0.213	1.219 ± 0.366	2.438 ± 0.320
Coil coating	0.610 ± 0.091	3.088 ± 0.351	0.610 ± 0.122	3.658 ± 0.381
Oil house paint	0.305 ± 0.137	3.962 ± 0.945	0.610 ± 0.213	4.877 ± 0.793

In laboratory-controlled environments, Spence et al.[36] studied the direct and synergistic effects of gaseous pollutants on commonly used exterior paints. Useful data were obtained on a vinyl coil coating, an acrylic coil coating, and an oil-base house paint. The oil-base house paint contained a calcium carbonate filler that was strongly attacked by SO_2 and moisture. The magnitude and variability in this effect masked any possible observed effect of ozone. Statistically significant effects of ozone were observed for the vinyl coil coating and the acrylic coil coating. There was a positive interaction effect between ozone and relative humidity on the vinyl coil coating, and a positive direct ozone effect on the erosion rate of the acrylic coil coating. Both of these coatings are very durable, which is why they were selected for factory application to aluminum siding material. Coatings as thin as 20 μm should last more than 20 years.

The vinyl coil coating was not affected by ozone at an input relative humidity of 50 percent, but it was at 90 percent. At the high relative humidity, the multiple regression coefficient for ozone was 0.00166 μm/year × (μg/m³). The average erosion rate in clean air was 1.3 μm/year.

A linear regression for the acrylic coil coating data gives:

$$\text{Erosion rate} = 0.159 + 0.000714\ O_3 \qquad (13\text{-}17)$$

where erosion rate is μm/year and O_3 is μg/m³.

Although the ozone effect on this coating is statistically significant, it has no practical significance because the erosion rate is so slow. At an average annual O_3 level of 100 μg/m³, this regression predicts that a 20-μm-thick coating would last over 80 years.

ECONOMICS

Most of the laboratory-observed effects of ozone on paint have little practical significance because their relative contributions to reducing useful life at ambient concentrations are so small. Ozone damage to some industrial maintenance paints and vinyl coil coatings could possibly produce economic losses.

Estimates of damage functions for the two affected coatings can be obtained by extrapolating the laboratory data to expected ambient conditions. The result for the industrial maintenance paint is:

$$\text{Erosion rate} = 1 + 0.01\ O_3 \pm 0.3 \qquad (13\text{-}18)$$

where erosion rate is mm/year, O_3 is μg/m³, and ±0.3 is the estimated standard deviation. Similarly, the result for the vinyl coil coating is:

$$\text{Erosion rate} = 1 + 0.001\ O_3 \pm 0.4 \qquad (13\text{-}19)$$

If erosion rate controls paint life, a percent life lost as a function of ozone concentration can be calculated. With estimates of the existing amounts of exposed painted surfaces and repainting costs, economic loss functions can be postulated.

Paint industry statistics are reported annually by Charles H. Kline and Co., Inc.[24] Estimated shipments of all industrial maintenance coatings in 1974 were 55 million gal. Alkyds that do not contain fillers probably account for 25 percent of those sales. Assuming a coverage of 300 ft²/gal at $0.40/ft² and half exterior exposure, the annual per capita repainting costs are estimated at $3.83. Considerable labor is involved in surface preparation and application of industrial maintenance paints.[14]

Coil-coated aluminum and glavanized steel have experienced a growth rate in shipments of approximately 13 percent from 1962 to 1974.[24] Seventy percent of these shipments are used in exterior applications. It is estimated from these production figures that 290 ft²/person of all coil coatings are exposed to exterior ambient conditions. If 40 percent are vinyl coatings and repainting costs are $0.25/ft², the per capita repainting costs for exposed susceptible coatings are $29. At a 23-year life expectancy, the annual cost is $1.26.

The fraction of paint life lost as a function of ozone concentration times the annual per capita repainting costs of affected paints gives an annual economic loss function. For industrial maintenance paints:

$$\text{Annual per capita costs} = \frac{0.0383\ O_3}{1 + 0.01\ O_3} \qquad (13\text{-}20)$$

And for the vinyl coatings:

$$\text{Annual per capita costs} = \frac{0.00126\ O_3}{1 + 0.001\ O_3} \qquad (13\text{-}21)$$

Industrial maintenance costs are indirectly paid by the public in higher prices for products and services. These costs are considered a nontaxable cost of doing business on which profit margins and markups are partially based. These difficult-to-quantify factors were not included in the per capita cost estimates as a function of ozone level. Lost tax revenues and markups could add considerably to the cost of ozone damage. Preventive maintenance is a nonproductive consumption of energy, labor, and materials that could be redirected to improve quality of life.

Effects on Other Materials

In a review of the effects of photochemical smog on materials, Sanderson[33] included possible effects on plastic tract recorders and asphalt. Because the effects were observed in laboratory tests at extremely high ozone levels (200,000 $\mu g/m^3$ and 104,000 $\mu g/m^3$, respectively), they should have no practical significance at ambient levels.

Haynie and Upham[20] reported a possible beneficial effect of photochemical oxidants on the corrosion behavior of steel. Their study used a nonlinear multiple regression technique to evaluate corrosion data as a function of field environmental conditions. Laboratory studies[19] did not show any statistically significant effect of ozone on steel corrosion. Some unmeasured factor that was covariant with photochemical oxidants could have caused the beneficial effect that was observed in the field studies.

SUMMARY

Although many organic materials have been shown to be susceptible to ozone attack, only damage to certain paints, elastomers, and dyes represents significant economic loss. Measures to prevent ozone damage to elastomers and dyes are a major cost. In contrast, the paint industry has not recognized the economic significance of possible ozone damage, and costs to prevent such attack are not identifiable.

Figure 13-1 graphically presents a summary of the estimated total annual per capita cost of ozone damage and preventive measures as a function of annual average concentration. The low and high estimates are based on data error and lack of confidence in many of the assumptions that were necessarily made.

This figure predicts that considerable savings from reduced damage to materials will be realized as the annual average ozone levels are decreased. This economic benefit is in addition to improved health, agriculture, visibility, and ecology, and thus it adds to the total quality of life.

Ozone materials damage as an individual problem can be solved more cost effectively by developing, identifying, and buying ozone-resistant materials. This approach has the distinct advantage of being applicable below natural background levels of ozone because, in general, there are no threshold limits to material damage.

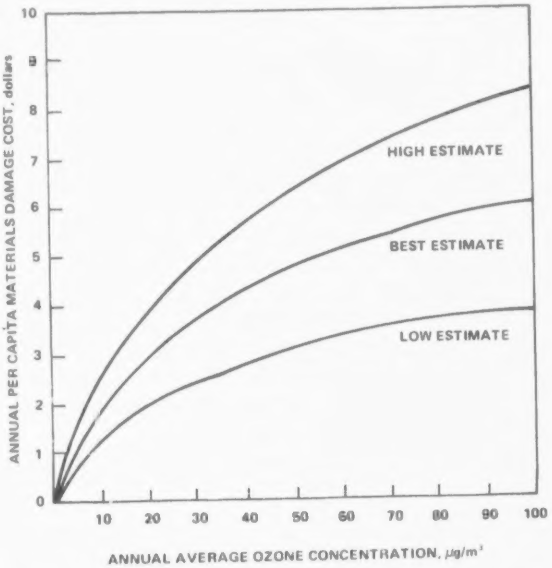

Figure 13-1. Effect of annual average ozone concentration on added costs resulting from damage to materials and preventive measures.

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